Shock Treatment: Efficacy, Memory Loss, and Brain Damage – Psychiatry's Don't Look, Don't Tell Policy

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Summary   Electroshock treatment, termed electroconvulsive therapy (ECT) by psychiatrists, is the induction of an artificial grand mal seizure in an individual by passing electricity through the brain. This paper addresses three aspects of this practice – its efficacy, its most salient effect, memory loss, and brain damage. In examining these issues it becomes apparent that psychiatry's policy has been to put a positive spin on dismal results, to limit research and investigation, and to tell the public as little as possible about the actual outcomes of ECT.   In other words, don't look, don't tell.

Introduction

In 2003, 2004, and 2005 I spent considerable time working as a paralegal for a law firm that was representing a man who had undergone ECT and subsequently lost virtually his entire autobiographical memory. After a course of 9 ECT treatments he no longer recognized his wife, children, or parents and lost most of his skill with computers and musical instruments. Although he was told his memory would return, it never did. A large part of my work involved reading published journal studies of ECT (approximately 200 studies covering a several decade period) and summarizing their major results for counsel. I also read many articles and books about ECT, including the task force reports on ECT published by the American Psychiatric Association.

Clearly I did not approach this issue as an impartial bystander. We firmly believed our client had been damaged by ECT and we set out to convince a jury that this had occurred.   However, while I can be fairly accused of approaching this subject from a less than neutral position, I would make several points in my defense.

It goes without saying that in order to argue against a particular position one must understand it. The defense in this case would obviously try to present ECT as a beneficial treatment, to offer evidence demonstrating that it was an effective and safe procedure. Thus, my readings of the literature had to focus on the positive as well as the negative. For purely pragmatic reasons it was necessary to consider any evidence that supported the use of ECT, not just the evidence of its damaging effects. We had to understand any pro-ECT arguments that opposing counsel might bring to trial.

Furthermore, studies which are critical of ECT are, as one might expect, seldom found in published journals. Those who study ECT and/or finance such research have a financial interest in the ongoing practice of ECT and are unlikely to pursue investigations or publish findings that might threaten ECT's continued existence. Contributing to the one-sidedness of the literature is

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the self-evident fact that opponents of ECT are not going to be involved in research that requires its use.

Thus, the deck is somewhat stacked against anyone looking for ECT's harmful effects in the medical literature. I state this only to point out that if I reached a negative conclusion regarding ECT, it was not because I "cherry picked" the negative studies and ignored the positive. The result would be a nearly empty "basket." The tendency in studies is to present ECT in a positive light. Virtually none of them conclude that ECT should not be practiced, that it is particularly harmful, or that the risk of damage outweighs the possible benefits.

On the other hand, a substantial percentage of individuals who have undergone ECT, not to mention a good many psychiatrists, are highly critical of the procedure and report serious debilitating effects. This clash of viewpoints is one that even the most independent of observers cannot fail to notice.

So where does the truth lie? To answer that question I was forced to look behind, beyond, and underneath the evidence presented by those who practice ECT and who, by reason thereof, are given money to study ECT and to publish results generally favorable to its practice. I was forced to ask what evidence had been swept under the research rug. What conclusions had been drawn that were not warranted by the evidence? What conclusions could be drawn from the evidence that were ignored? What research should have been done that never was done? If a psychiatrist says, as one did, that there are no published journal studies saying ECT causes permanent memory loss, is that because exhaustive research has proven such an effect does not occur, or because no thorough investigation was ever undertaken? Or is he simply ill informed – or lying?

This paper presents some of the information I uncovered in posing these questions and others. I ask only that the reader consider the evidence I present and decide whether my reasoning is sound. I believe that few, if any, of the individuals who chose electroconvulsive therapy do so with full informed consent. Indeed, I am convinced that their consent is utterly misinformed. It is my hope that this paper will explain why.

Foreword

Built into a discussion of most subjects within the field of mental health is a certain semantic "Catch-22." The very language one must use in talking about mental and emotional dysfunction, including the term "mental health" itself, perpetuates a psychiatric worldview that is primarily a marketing fiction.

Those conditions that psychiatrists call mental illnesses have never been proven to be illnesses. Indeed, the weight of evidence on this question fails to support the brain disease theories. According to Elliot Valenstein, University of Michigan Professor Emeritus of Psychology and Neuroscience, "… the evidence and arguments supporting all these claims about the relationship of brain chemistry to psychological problems and personality and behavioral traits are far from compelling and are most likely wrong."[1] Harvard psychiatrist Joseph Glenmullen discussed this issue in his book, *Prozac Backlash*. He wrote,

> In recent decades, we have had no shortage of alleged biochemical imbalances for psychiatric conditions. Diligent though these attempts have been, not one has been proven. Quite the contrary. In every instance where such an imbalance was thought to have been found, it was later proven false.[2]

The U.S. Surgeon General's 1999 report, *Mental Health: A Report of the Surgeon General – Executive Summary*, referred to the "lack of objective, physical symptoms" in mental disorders.[3] and stated, "…there is no definitive lesion, laboratory test, or abnormality in brain tissue that can identify the illness."[4] Last year the President of the American Psychiatric Association (APA), Steven Sharfstein, admitted, "We do not have a clean-cut lab test" to detect chemical imbalances.[5]

Nevertheless the idea that certain emotions and behaviors are the result of this undetectable "illness" persists and has, in fact, become imbedded in our language. It is difficult, if not impossible, to discuss anything in the mental health field, including ECT, without using such words as "patient," "treatment," "therapy," and "symptom"- thereby giving implicit agreement to an unproven dogma that is not supported by medical science.

I am therefore bound to warn readers of this state of affairs and hope that they will resist the subliminal indoctrination inherent in this pathological lexicon – double entendre intended. It is the skill of the snake-oil huckster to get the normal person to think of himself as a "patient" in need of "treatment." Historically, what psychiatrists have called therapies – lobotomy, ice water immersion, various types of restraint, to name a few - are more properly termed torture. The reader should therefore be on guard, understanding that if I use the language of psychiatry to discuss ECT, it may only be to avoid unnecessary clumsiness in discussing this subject. It has never been shown that psychiatric disorders are illnesses and the fact that psychiatrists have invented diagnoses that can be used to assign individuals the identity of a sick "patient" does not mean there is any validity to that process of stigmatization.

I would also note that the use of the terms electroconvulsive therapy and ECT, rather than shock or shock treatment, in the following work, is intentional. I have chosen to accept psychiatry's term for this procedure so that the evidence I present is not clouded by any language that might be considered prejudicial. The published facts speak loudly and clearly enough for themselves.

The Effectiveness of ECT

Electroconvulsive therapy is often claimed to be psychiatry's most effective treatment. One textbook on psychiatry written for medical students calls it "one of the most humane and most efficacious treatments available in mental health care."[6] The evidence suggests otherwise.

The efficacy of ECT can be defined in terms of two fundamental factors: response rate and relapse rate. Response rate refers to the percentage of individuals who achieve a significant reduction in their depression after undergoing ECT. Relapse rate is essentially a measurement of how long the response lasts. How many patients are still substantially free of depression a week later? A month later? How long before they relapse and return to being depressed?

The most commonly used test for measuring the degree of depression is the Hamilton Rating Scale for Depression (HRSD), a 17-question test in which the subject is asked about his or her mood, suicidal thoughts, feelings of guilt and anxiety, sleeping and eating patterns, and other possible "symptoms." Answers may be assigned a value anywhere from 0 to 4, with four indicating that the symptom is severe. Scores on the HRSD-17 can range from 0 to 52. The depression is considered mild if scores range from 10 – 13, mild to moderate in the 14 to 17 range, and moderate to severe in the 18 and over range.

Psychiatrists typically assert that 70 – 80% of patients respond favorably to ECT. But what does this really mean? ECT studies differ in how they define response, and in some cases a single study may employ two different criteria, one strict and one more lenient, in judging whether or not a patient has responded. Definitions of response involve three related factors: 1) the percentage drop in the HRSD score, 2) how low the final score is, and 3) how long the response lasts (i.e., how long the score remains low). Knowledge of the definition of response used in ECT studies is critical to understanding what is actually meant by a 70% response rate and whether such figures accurately convey the effectiveness of this treatment.

Response Rate

A 1990 study by Joan Prudic used two response criteria. Criteria A defined response as a minimum 60% reduction in the Hamilton Rating Scale for Depression (HRSD) score, resulting in a score of 16 or less. Additionally, Criteria A required that the 60% reduction last at least one week. Criteria B differed from criteria A in requiring a final HRSD score of 9 or less.[7]

Thus, the patient who entered Prudic's 1990 study with an HRSD score of 30, had to achieve an 18 point drop in the HRSD score (i.e., a final score of 12) and that drop had to persist for at least a week for the patient to be considered a responder. But for a patient who entered with a score of 32, an 18 or even 19 point drop (final score 13 or 14) would not be sufficient, even though the post-ECT score was less than 16, since even a 19 point drop would represent only a 59.4% reduction in the HRSD score.

(One might ask whether a hidden source of bias might enter into the post-ECT testing, since HDRS evaluators would want subjects to score as low as possible to achieve the 60% reduction and one point could make the difference. I do not know whether this possible source of bias has ever been studied. The studies I read gave no indication that HDRS evaluators were kept blind to the subjects' pre or post-ECT status.)

Another Prudic study, published in 1996, defined response as a post-ECT score of HRSD score of 10 or less. (The mean pre-ECT score for patients in this study was approximately 30.) Patients were tested immediately after (within 3 days) and a week after (6-8 days) ECT cessation. Response rates were presented for both test conditions.[8]

A 2000 Sackeim study used "final responder" criteria identical to Prudic's criteria A and "final remitter" criteria requiring an HRSD score of 10 or less one week after ECT.[9]

A 2004 Prudic study employed both a "moderate criteria" for remission that required HRSD scores of 10 or less immediately after the ECT course, and a "strict criteria" that required a post-ECT score of 7 or less. In explaining her strict criteria Prudic cited 3 previous studies which had argued that "an HRSD threshold of 10 might allow for significant residual symptoms."[10] In other words, an individual with a score of 10 might still be thought of as having significant symptoms of depression.

The definition of response or remission used in a study impacts the rate of response or remission found. An HDRS test given immediately after ECT is likely to result in a lower score than a test given a week later, since depression tends to begin returning almost immediately after the course of treatments (more about this later). Of course the maximum score allowed (16, 10 or 7) also affects the response rates measured: the stricter the criteria, the fewer the number of subjects who will be found to have remitted.

The importance of response definitions is illustrated by the following statement taken from the 2001 American Psychiatric Association (APA) task force report on ECT:

> Among patients who are receiving ECT as a first-line treatment or who have received inadequate pharmacotherapy during the index episode because of medication intolerance, response rates continue to be reported in the range of 80%-90% (Prudic et al. 1990, 1996). Among patients who have not responded to one or more adequate antidepressant trials the response rate remains substantial, falling in the range of 50%-60% (Prudic et al. 1996; Sackeim et al. 1990b, 2000).[11]

The phrase "inadequate pharmacotherapy" requires a brief explanation. The adequacy of the drug treatment that precedes ECT is evaluated primarily on the basis of the dosage of the drug taken and the length of time the drug was taken. If a patient has taken a high enough dosage for a sufficient length of time the treatment is considered "adequate." In Prudic's 1996 study, for example, a patient who had taken at least 200 mg/day of imipramine (Tofranil) or 20 mg/day of fluoxetine (Prozac) for at least 4 weeks was considered to have received adequate pharmacotherapy. Equivalent criteria for other antidepressant drugs also qualified as adequate treatment.

Subjects who fail to respond to "adequate" drug treatment are called "medication resistant" – a rather disingenuous term when applied to antidepressants - and psychotropic medication in general - since it implies that 1) antidepressant drug treatment is usually successful and 2) when it doesn't work, there is some unusual patient resistance involved. Both of these implications are false. The truth is much simpler: most subjects who take antidepressants are soon going to qualify as medication resistant because the effectiveness of antidepressants is quite minimal.[12]

The APA task force cites two Prudic studies in claiming ECT response rates of 80%-90%. It is true that the 1990 study did find a response rate of 86% for individuals with inadequate pharmacotherapy. However, Prudic's 1990 study, as we noted above, used two criteria for response. The 86% response rate was for those who met criteria A, which required a final HDRS score of 16 or less – a relatively high cut-off point, particularly considering that individuals with a score of 10 are viewed by some researchers as manifesting significant symptoms of depression. The strict criteria (criteria B) measured the response rate for patients who had received inadequate pharmacotherapy at 69% - not 80%-90%.[13] Prudic's 1996 study measured response rates of 91.4% immediately after ECT but a week later that figure was down to 74%.[14] Clearly the task force decided to present the most favorable statistics.

This positive spin is then presented to the public, while the 50%-60% statistic cited for individuals who have not responded to an adequate trial of an antidepressant is left buried in the task force report, even though the use of ECT has always been justified on the basis of "medication resistance" – i.e., treatment with antidepressants failed.

The APA report itself states, "ECT is most often used in patients who have not responded to other treatments."[15] Whether this is actually true was recently called into question by an article in *The Journal of ECT* which, while noting, "Resistance to antidepressant medication is the commonest stated indication for electroconvulsive therapy by psychiatrists," also found that, among 37 patients who had been referred for ECT due to medication resistance, "only half the sample met contemporary criteria for medication resistance." The study also pointed out that "no agreed-upon operational definition for resistance to antidepressant medication exists."[16]

But regardless of whether *most* patients referred to ECT have failed an adequate trial of antidepressant medication or whether medication resistance is merely a convenient justification for using ECT, research has shown that previous treatment with antidepressants can have a significant impact on response to ECT and there is no doubt that many, if not most, patients referred for ECT have experienced at least one and in many cases more than one adequate trial of an antidepressant.

If, as the APA task force states, most individuals referred for ECT "have not responded to other treatments," then one might expect the APA and practitioners of ECT to make it very clear to patients and public alike that a large percentage of patients have only a 50%-60% chance of responding.

This is not done.   Instead, those who advertise ECT services frequently claim success rates as high as 90% and stay away from specific information about the impact of previous drug treatment on a patient's chances of responding to ECT.

The following claims are typical of what the public is told regarding ECT's effectiveness.

> Response rates for an uncomplicated depression can be as high as 90%. For refractory depressions (those that haven't responded to conventional medication treatment), the response rate is still in the 70-80% range in many studies.[17]

> Some studies show an 80% response rate for treating major depression with ECT.[18]

> Electroconvulsive therapy is between 60% and 90% effective in the treatment of major depression.[19]

> … ECT helps patients about 80% of the time….[20]

These statements are misleading on a number of levels. One key missing element is the definition of "response" that we have just discussed. Prospective patients are surely not going to be aware that the words "response", "effective," or "helps," as defined in ECT research, refer to effects that may last only a few days. They would naturally assume that "response" meant an improvement that was maintained for more than a week.

Nor would patients be aware that the definitions of response are so varied – that one study's responsive patient would be another study's still depressed patient.

Finally, as we discussed above, patients would be unaware that, if they have had at least one adequate trial of an antidepressant, their chances of responding may be much closer to 50% than 80% - and, again, that this "response" has only been confirmed to last for a week.

The current (2006) APA website contains the following statement: "Clinical evidence indicates that for uncomplicated cases of severe major depression, ECT will produce a substantial improvement in 80% of patients (1). ECT has also been shown to be effective in depressed patients who do not respond to other forms of treatment (2)."[21] The second part of this statement, though it refers to medication resistant patients, contains no statistic, since that statistic would have had to have been 50%-60%. The APA here exhibits its characteristic selectivity in citing studies to back up its claims. The references cited for this statement – a 1988 study by Weiner (1) and a 1990 study by Prudic and Harold Sackeim (2) - conveniently ignore more recent studies that are much less encouraging.

One of those recent works, also by Prudic and Sackeim and published in 2004, looked at the outcomes of ECT as it is practiced in the community. Although a 50%-60% response rate is certainly a more realistic outcome expectation for most ECT subjects, this rate is itself misleading, since it is based upon controlled studies in research settings. The vast majority of patients are not part of such studies.

So what are the remission rates for those who receive ECT in a community setting? Prudic and Sackeim found that, "In contrast to the 70%-90% remission rates expected with ECT, remission rates, depending on criteria, were 30.3%-46.7%."[22] The study concluded, "The remission rate with ECT in community settings is substantially less than that in clinical trials."[23]

30.3% is a long way from 90%. But even this figure is overly optimistic. Remission in this study was measured, on average, within 3 days of ECT termination, "with 318 of 347 patients (91.6%) evaluated within 10 days."[24] According to the study, "A longer interval to assessment was associated with less improvement and lower rates of response and remission."[25] Thus, if all the patients had been measured at least one week post-ECT, the remission rates would have been even lower. The study found, "...on average, 10 days after ECT, patients had lost 40% of the improvement that accrued over the ECT course."[26]

Relapse Rate

Our examination of the concept of a response rate has inevitably led us to a second closely related factor: How long does the "response" last? How soon after ECT does the patient relapse? Prudic's 2004 community study found that after 6 months, only 22.5% of patients met the moderate remission criteria and 16.1% met the strict criteria,[27] with most patients relapsing within 3-4 months.[28]

We have moved from the APA's rosy 90% rate of effectiveness to a much more realistic 16%, a rate that conceivably could be matched or bettered by placebo treatment alone.

As dismal as these 2004 figures are, they should have come as no surprise to anyone. In 1990 Prudic had measured response rates for patients who had received adequate pharmacological treatment pre-ECT ("medication resistant" patients) between 42% and 50% depending on the strictness of the response criteria used.[29] Prudic had also suggested, "The findings may underestimate the importance of medication resistance in predicting ECT response."[30] In another 1990 study, Sackeim found that 64% of medication resistant patients relapsed within one year, with nearly all those relapses occurring in the first four months.[31] In 1991 Devanand, Sackeim and Prudic, citing their earlier 1990 studies, warned, "…the detrimental impact of medication resistance on both the acute response to ECT and the propensity to relapse may be even greater than that reported in these two studies."[32]

Thus, by the early 1990's it was known that most patients who are referred for ECT, patients who often have long histories of drug treatment, have only a 50% chance of responding and at least a 50%-60% chance of relapsing soon after ECT, resulting in a true remission rate of 20-25% a few months post-ECT, similar to what Prudic found in 2004.* And these figures, as both

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* One might speculate that, if ECT's antidepressant effect is a result of the brain's response to ECT's electrical assault, perhaps what is being called a result of "medication resistance" is actually the result of damage to the brain's ability to "defend" itself caused by antidepressant and other medications.

Prudic and Sackeim suggested, were quite possibly underestimates. It is doubtful that this information ever reached the public.

Another Sackeim study, published in 2001 in the *Journal of the American Medical Association*, confirms these figures. In that study 290 patients completed a course of ECT. Only159 (55%) were considered to be in remission 4-8 days post-ECT. This represents an initial 45% failure rate. 84 patients participated in the second phase of the trial. They were divided into three groups. One group received placebo, one group received continuation pharmacotherapy with nortriptyline, and the third group received nortryptyline and lithium. The study found, "Over the 24-week trial, the relapse rate for placebo was 84% (95% confidence interval [CI], 70%-99%); for nortriptyline, 60% (95% CI, 41%-79%); and for nortriptyline-lithium, 39% (95% CI, 19%-59%) …. Medication-resistant patients, female patients, and those with more severe depressive symptoms following ECT had more rapid relapse."[33] The relapse rates were "72.2% for nonpsychotic medication-resistant patients (n=36)"[34] and the authors concluded that "… without active treatment, virtually all remitted patients relapse within 6 months of stopping ECT."[35]

It should be mentioned that the authors of this study considered their remitter criteria for the first phase of the study to be quite stringent. Patients were defined to have remitted only if they had a 60% reduction in HRSD scores and a "maximum HRSD score of 10 at both at an assessment within 2 days of ECT discontinuation and reassessment 4 to 8 days following ECT termination, while free of psychotropic medication."[36] Less stringent criteria might have produced higher remission rates initially but the authors were aware that "the extent of residual symptoms is predictive of relapse following antidepressant treatment."[37] They wanted the participants in the second phase of the study to begin that phase with as few "residual symptoms" as possible, giving them the best chance of remaining in remission.   So even though the study was designed so that participants in the second (post-ECT) phase of the study would be those with the best chance of succeeding (i.e., not relapsing), the results were dismal.

One way to look at this is to suppose that all 159 patients who remitted were put on placebo post-ECT. 134 would relapse within 6 months, leaving 25. If 290 patients were given ECT and not returned to drug treatment, only 25 out of 290 (9%) would not still be depressed six months later – and most of them would have relapsed in 3-4 months. The most depressed patients would relapse much earlier. All of this begs the question of whether a similar result could be obtained by doing nothing. Sackeim himself admitted, "… almost universal relapse should be expected without effective continuation therapy."[38]

This study shows that ECT is likely to fail virtually all of the seriously depressed individuals to whom it is given. They are depressed and taking antidepressant drugs before ECT and they must continue taking drugs post-ECT with minimal prospects for improvement in their condition.

The sham ECT studies

In short, ECT is extraordinarily ineffective. The American Psychiatric Association admitted as much in its 2001 task force report on ECT when it discussed the sham ECT studies. (In the sham ECT condition, the patients receive a general anesthetic, electrodes connected to the ECT machine at attached to their heads, and the button that would normally deliver current is pushed. However, no current is delivered. The patients do not know whether real or sham ECT was delivered. The evaluations of the patients' responses using the HRSD or other tests are done by psychiatrists who do not know whether a given individual received real or sham ECT.)

According to the report,

> Studies prior to 1980 failed to demonstrate a therapeutic advantage of real ECT relative to sham treatment[*] (Brill et al. 1959a, 1959b, 1959c; Heath et al. 1964; Miller etal. 1953). In contrast, three later studies all found a substantial advantage for real ECT in short-term therapeutic outcome (Abraham and Kulhara 1987; Brandon et al. 1985; Taylor and Fleminger 1980).… All of the recent studies involved the use of antipsychotic medications in both the real ECT and the sham groups.…"[39]

It is worth noting that all three studies cited by the task force were studies of ECT in the treatment of schizophrenia. None was a depression study. All used antipsychotic drugs during the treatment course, an obvious confounding factor. In both the Abraham and Kulhara and Brandon studies the superiority of real ECT over sham ECT disappears by 8 weeks post-ECT.[40] The Brandon study also found no significant differences between the real and sham ECT groups on the Hamilton Depression Rating Scale at the conclusion of the treatment course (4 weeks).[41] The Taylor and Fleminger study rated the ECT group as only slightly improved over the sham group at 4 weeks post-ECT. They did not test at 8 weeks but found that "by 16 weeks [12 weeks post-ECT] there was little difference between the two groups."[42]

For the APA's own task force to not cite a single study in which real ECT demonstrated a superior outcome over sham ECT in the treatment of depression is remarkable. ECT is, of course, used principally in the treatment of depression.

Also of significance is the failure of the task force to mention, in its discussion of real vs. sham ECT, Brandon's 1984 study of real vs. sham ECT in the treatment of depression. That study only found an advantage for real ECT during the course of ECT itself. "At follow up at 12 and 28 weeks there was no difference between the treatment groups."[43] This is a post-1980 study which found real ECT to have no advantage over sham ECT, except during the course of ECT itself, when the patient may well be confused and disoriented.

Moreover, these results, as far as depression is concerned, were replicated in Brandon's 1985 schizophrenia study, which, as noted above, also found no significant differences in depression ratings between sham and real ECT at ECT termination. The task force was silent regarding the real implications of both of Brandon's post-1980 studies.

Effectiveness and EEG – ECT's adverse neurophysiological effects

There is no doubt, however, that ECT does *something*. Passing electricity through the brain and causing a seizure, and doing this two or three times a week for several weeks, is obviously going to produce profound effects. To fully understand what ECT does and how it might be producing its alleged "antidepressant" effect, some familiarity with the neurophysiological effects of ECT, particularly its effect on brain waves*, is necessary.

Psychiatrist Andrew Krystal, another frequently-cited ECT researcher, discussed ECT's impact on brain waves in a 2000 paper. Krystal wrote,

> The most pronounced change in the EEG during a course of ECT is an increase in the amount of slow activity (activity that is less than 8.5 Hz in the waking EEG). It [the slowing] is largely gone by 1 month after the ECT course, and is only rarely present past 3 months.
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> A decrease in the average EEG background frequency and in the amount of higher frequency beta activity have also been reported to occur with ECT; however, these effects are of uncertain clinical significance [Fink, 1979; Weiner, 1983].[44]

Sackeim reviewed previous findings of ECT's adverse neurophysiological effects in a 2000 study.

> During the postictal [after a seizure] period and for a period of weeks following the ECT course there is a prominent increase in slow-wave activity (delta and theta) in the electroencephalogram (EEG) (Fink and Kahn, 1957; Weiner, 1983; Sackeim et al., 1996.[45]
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> We have also reported that increased delta power over prefrontal sites in the interictal [relating to the period between seizures] EEG is positively associated with therapeutic response (Sackeim et al., 1996). The latter finding agrees with earlier observations that increased delta activity is linked to efficacy (e.g., Fink and Kahn, 1957; Roth et al., 1957; Ottosson, 1962; Strömgren and Jensen, 1975), and provided evidence of a specific topographic distribution for this effect.[46]

Sackeim's 2000 EEG study found that "increased theta activity in left frontotemporal regions was associated with longer duration of disorientation"[47] and "increased theta power in left frontotemporal regions was associated with greater retrograde amnesia for autobiographical events.[48] This supports Sobin's 1995 finding linking the length of disorientation after ECT treatments with the magnitude of retrograde amnesia suffered by the subject.[49]

Thus, we see that ECT produces increases in slow wave activity (delta and theta waves) and decreases in higher frequency beta activity during the ECT course. Delta waves are present in

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* Brain waves are recorded on an electroencephalogram (EEG), a graphic recording of the ongoing electrical activity generated by neurons in the brain. This activity is divided into various frequencies. The unit of frequency used is the hertz. One hertz is defined to be one cycle per second. The lowest frequency waves are delta waves, 3 Hz or below. Theta waves range from about 3.5 to about 7.5 Hz.. Alpha waves are between 7.5 and 12.5 Hz. Beta waves have a frequency of 12.5 Hz or greater.

deep, dreamless sleep. Theta waves are found in the early stages of sleep and are associated with daydreaming and fantasizing. Higher beta waves are associated with thinking, decision making, and awareness of self and surroundings. ECT appears to lower awareness and increase unconsciousness from the point of view of EEG data, with this altered state of consciousness continuing for approximately four to twelve weeks. According to Sackeim, " …increased delta and theta power can be more readily interpreted as reflecting adverse neurophysiological effects relative to alterations in the alpha or beta frequency bands (Ball et al., 1977; Petsche et al., 1984; Steriade et al.,1990; Pedley and Traub, 1990)."[50]

ECT's adverse neurophysiological effects would appear to put patients into a sort of gradually diminishing sleep/fantasy state for several weeks to months. Most experience relapse or a return of depressive symptoms within that time. We recall Prudic's 2004 study (discussed above) which found that 40% of the "improvement" following ECT was lost within 10 days. The finding of no difference in outcomes between sham and real ECT at 4 to 8 weeks also bears upon this point.

More intense adverse EEG effects, greater "therapeutic response," and greater cognitive side effects appear to form an ECT trio. Krystal writes,

> Given the well-known relationship between the presence of slow-wave activity in the waking EEG and encephalopathy, as well as reports of greater EEG slowing from types of ECT treatment associated with greater cognitive impairment (sine-wave and BL* ECT), it is surprising that relatively few studies have examined the relationship between interictal EEG slowing and the cognitive effects of ECT [Weiner, 1983; Krystal and Weiner, 1999]. Several studies suggest that greater EEG slowing appears to be associated with greater cognitive side effects with ECT. Earlier studies reported that greater EEG slowing was associated with greater memory loss with ECT.[51]

The question naturally arises: Is the alleged "therapeutic" effect of ECT at least in part the result of its adverse effect on brain waves? Krystal infers as much when he writes,

> …seizures having a more intense and more widespread physiologic effect on brain function are associated with a better therapeutic response but also greater cognitive side effects.[52]

Bilateral (BL) ECT*, as Krystal stated above, is associated with greater EEG slowing. It is also considered to produce a better therapeutic response than unilateral treatment, unless that treatment is delivered at high multiples of the subject's seizure threshold. (The seizure threshold is essentially the amount of current that must be delivered to produce a seizure that lasts at least 25 seconds.) In the May 2000 issue of the *Archives of General Psychiatry*, Sackeim reported

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* In bilateral (BL) ECT, the electrodes that deliver the charge to the brain are placed on the temples, one on each side of the head. In unilateral ECT one electrode is placed on the temple (usually on the right) and one, roughly speaking, in the middle of the top of the head.

that unilateral ECT at six times (500% above) the seizure threshold was as effective as bilateral treatment at 2.5 times the threshold.[53] Right unilateral (RUL) ECT at only 150% above threshold was much less effective.

Of course, increasing the electrical dosage to six times threshold increases the "intense and more widespread physiologic effect on the brain" to which Krystal refers:

> There is strong evidence that higher intensity ECT results in greater global (transcortical) slow wave activity (Fink et al. 1958; Ottoson 1960; Proctor & Goodwin 1943; Robin et al. 1985; Weiner et al. 1986b)…. … such global EEG dysfunction may reflect dosage effects in deep structures.[54]

That increased intensity is predictably accompanied by greater cognitive deficits. In the same May 2000 issue of the *Archives of General Psychiatry*, McCall, Rebousin, Weiner, et al. (including Sackeim) reported the results of a study of right unilateral ECT. They found, "… the likelihood of both antidepressant response and cognitive deficits increased as stimulus dose increased relative to initial seizure threshold, up through 8 to 12 times the threshold."[55]

The known associations are as follows:

1. ECT causes increases in slow waves and decreases in beta waves.
2. Increases in slow (delta) waves are associated with therapeutic response and efficacy.
3. Increases in slow (theta) waves are associated with longer disorientation and worsened amnesia.
4. Bilateral treatment and high dose treatment are both associated with longer disorientation and more extensive amnesia.
5. Bilateral treatment is associated with greater EEG slowing.
6. Bilateral treatment is associated with greater "effectiveness."

These associations support an effectiveness-brain dysfunction link. In a 53-page paper published in *Behavioral and Brain Sciences*, ECT expert Richard Weiner cites a study which found EEG abnormalities were "15% at 1 month post-ECT, 6% at 2 months, 2% at 3-6 months, and 1% at 1 year."[56] Based upon these findings alone, if ECT's effectiveness is the result of EEG abnormalities, one might expect 15% of ECT patients to experience a "therapeutic" effect lasting at least one month, with 85% relapsing during that time period, if given only placebo post-ECT. This is rather close to Sackeim's relapse rate of 84% of patients on placebo (study cited at footnote 29). That relapse rate is, admittedly, over 6 months. But the majority of Sackeim's patients relapsed earlier, within Krystal's 1 – 3 month window for EEG abnormalities. 50% of Sackeim's placebo patients relapsed in the first week post-ECT. Nearly 70% had relapsed by three months. According to Michael Alan Taylor, professor of psychiatry at the Chicago Medical School's Finch University of Health Sciences, "The largest number of relapses occur in the first 10 days after a successful course of ECT, with the majority of the remaining relapses occurring during the next 5 weeks."[57]

The difference between our predicted 85% relapse rate, based on EEG results alone, and lower relapse rates found in various studies may be due to other biochemical or neurological

effects that keep the brain (not to mention the psychological condition of the patient) in an abnormal state post-ECT. The placebo effect may also play a role. These factors might contribute to a lengthened time period of apparent "antidepressant" effect. But EEG abnormalities certainly appear to play a significant role in the "antidepressant" effect of ECT. As these abnormalities wear off and the brain returns to normal, the depression returns. The sham ECT studies, as well as the data on relapse rates, suggest that real ECT loses any advantage over sham ECT as soon as the brain abnormalities subside. (For an excellent analysis of the sham ECT literature I would refer the reader to psychiatrist Colin Ross's paper, "The Sham ECT Literature: Implications for Consent to ECT," *Ethical Human Psychology and Psychiatry*, 8, 17-28, 2006.)

Link between therapeutic and adverse cognitive effects

As we have seen, ECT's alleged "therapeutic" effect appears to be closely linked to its adverse cognitive effects. The idea that ECT "works" by damaging the brain (regardless of the brain's ability to repair the damage) is one that psychiatrists who practice ECT would rather minimize or deny.   In his 2000 EEG study Sackeim stated that he found no such relationship.

> There were no significant relations between degree of clinical improvement and changes in global cognitive status (MMS) ($r = 0.08$, $df = 53$, NS) or with retrograde amnesia scores ($r = 0.08$, $df = 45$, NS).   …the magnitude of cognitive effects was independent of clinical change.[58]

But the link between cognitive effects and clinical change cannot be so easily dismissed. We already discussed McCall's study which found a relationship between antidepressant response and cognitive deficits up through 8 to 12 times seizure threshold.

Sackeim's 2000 EEG study itself provides more evidence. Table 2 in this study reveals what occurred as the treatment conditions changed from the least to the most effective - from low dose right unilateral (RUL) ECT, to high dose RUL, to low and high dose bilateral (BL). The duration of post-ictal disorientation (see below for a discussion of this effect) grew substantially longer, the modified Mini Mental State exam scores grew significantly worse, and retrograde amnesia for autobiographical information also grew much worse.   Sackeim wrote, "High-dose RUL ECT resulted in more prolonged orientation recovery than low-dose RUL ECT (p < 0.05), while orientation time was equivalent in the two BL ECT conditions and *prolonged relative to either of the RUL ECT conditions* (p≤0.0002)."[59] (emphasis added)

It is clear from this study that the higher dose RUL condition produced more cognitive dysfunction than the lower dose RUL condition and that bilateral treatment produced considerably more disruption than either RUL condition.   Also, the high dose RUL condition in this study was only 150% above threshold. This is significant because, as we noted above (p. 14, top), RUL ECT at 150% above threshold was found to be much less effective than bilateral ECT. Thus, although Sackeim does not present the data on clinical change for the various treatment groups, we can reasonably conclude from this study that the most "effective" forms of treatment (bilateral and high dose as opposed to unilateral and low dose) also produced the most cognitive dysfunction.

Additional evidence for a link between ECT's "antidepressant" effect and its cognitive impact comes from Sobin's 1995 study, which found, "Longer duration of acute disorientation was also associated with greater persistent retrograde amnesia."[60] Sobin also found, "Long-term retrograde amnesia was significantly influenced by whether or not patients had received a second, crossover course of ECT."[61] The second "crossover" course of ECT is a course of high dose bilateral ECT given to subjects (most often those assigned to the unilateral treatment condition) who did not respond to the first ECT course. The crossover course is given because it increases the chance of a "response" where none has yet occurred. But this response, this "effectiveness," clearly comes at a price – greater persistent retrograde amnesia.

A 2003 meta-analysis by the UK ECT Review Group (led by Oxford's John Geddes) concluded,

> In general there seems to be a positive relation between the amount of electrical current administered to the dominant hemisphere and both the clinical efficacy and the amount of cognitive impairment caused by ECT.
>
> Therefore there is a trade-off between making ECT optimally effective in terms of amelioration of depressive symptoms and limitation of cognitive impairment.[62]

We should also mention Douglas Cameron's excellent analysis of the history of ECT machines.[63] Cameron shows that when the manufacturers of ECT machines produced devices that used a minimal amount of current to induce seizures, with the goal of reducing memory damage, the result was less effectiveness. This was followed by a return to the manufacture of more powerful devices - ones that could deliver higher doses of electricity to the brain. In 2000 several psychiatrists called for machines that could deliver even higher electrical charge to the brain "to ensure adequate treatment response."[64]

Post-ictal disorientation

Disorientation post-ECT is one of the cognitive effects of ECT. A strictly analytical approach to ECT, as we are pursuing here, can numb us to the human aspect of what we are discussing. The subject of disorientation provides an opportunity to reacquaint ourselves with ECT's real impact, while at the same time observing again the link between "effectiveness" and cognitive deficits. Disorientation was defined in Sackeim's 2000 EEG study as a subject's inability (after an ECT treatment and once the eyes had opened) to provide a questioner with at least four of these five pieces of information: their name, age, date of birth, current location, and day of the week.

The duration of post-ictal disorientation in subjects who received low-dose RUL ECT averaged 10.8 minutes, with a standard deviation of 8.3 minutes. High-dose RUL ECT resulted in disorientation lasting a mean of 21.2 minutes, with a standard deviation of 11.8 minutes. The corresponding mean and standard deviation figures for disorientation for low and high dose bilateral ECT were 39.4 (SD = 20.9) and 42.7 (SD=16.0), respectively.[65] (In a normal distribution, about 68% of the scores are within one standard deviation of the mean and about 95% of the scores are within two standard deviations of the mean.)

Sackeim wrote, "If recovery of orientation did not take place within 90 minutes, patients were given a score of 100 minutes."[66] It would appear then, that by shrinking the upper end of these scores, the means were artificially lowered. In any case, the bilateral conditions resulted in a period of disorientation lasting four times longer than that of the ineffective low dose RUL condition. There is no doubt that a significant number of BL subjects were, for periods of well over an hour, only able to answer correctly three or fewer of the orientation questions.

What sort of impact on the brain does it take to create such confusion – a mental state in which people don't know where they are, or remember their date of birth, for a period of over an hour, even though their eyes are open and they are at least partially conscious?   This is, to a certain degree at least, a rhetorical question. But there is value in balancing our strictly analytical viewpoint with one that takes a more common sense approach and simply asks whether the brain should be subjected repeatedly to this sort of impact.

Let's not forget that the brain actively works to stop the ECT's effect from continuing or occurring in the future – another one of those "common sense" points that are worth keeping in mind.   In the 1994 paper referenced above Sackeim wrote,

> Seizures do not terminate because of inadequate carbohydrate supply, neuronal Exhaustion, or other passive processes. Indeed, the fact of status epilepticus [prolonged seizures] indicates that the brain is capable of sustaining seizure activity for days….

> It is established that ECT has powerful anticonvulsant properties (Sackeim et al. 1986a). These include the progressive increase in seizure threshold that occurs during the ECT course, the progressive decrease in seizure duration, the regional and/or global reductions in CBF [cerebral blood flow] and CMR [cerebral metabolic rate], and the induction of slow wave activity in the EEG.[67]

 Whether ECT "works" by producing cognitive deficits, i.e., whether the short term "therapeutic" effect is the direct result of cognitive dysfunction, may be difficult to prove. But it is, in one sense, irrelevant.   There is no doubt that ECT's effect on mood is accompanied by widespread and powerful adverse physiological effects on brain function and cognitive dysfunction.

Continuation ECT

Psychiatry's "solution" to ECT's lack of effectiveness is often to keep the patient in a state of perpetual brain dysfunction with continuation (weekly or monthly) ECT. The 2001 APA task force report states,

> Although psychotropic continuation therapy is the prevailing practice, few studies document the efficacy of such treatment after a course of ECT. Even in patients complying with such regimens, some recent studies report high relapse rates [5 citations]. These high relapse rates have led some practitioners to recommend continuation ECT for selected individuals [4 citations]. Recent reviews have tended to report surprisingly low relapse rates among patients receiving such treatment [9 citations]."[68]

The probable results of such practice are suggested in the following quote from the 2001 APA task force report:

> Owing to a combination of anterograde and retrograde effects, *many patients* may manifest persistent loss of memory for some events that transpired in the interval starting several months before and extending to several weeks after the ECT course [emphasis added].[69]

Weekly and monthly ECT could then be expected to result in a temporal window of memory loss stretching for years. But as we shall see, it doesn't take continuation ECT to produce such an effect. It occurs with one series of shocks.

Memory Loss and ECT

Memory loss is perhaps the most discussed side effect of passing electricity through the brain. The degree and permanency of such memory loss has been a topic of some debate within the psychiatric profession. Various studies have addressed this issue. Many have concluded that memory loss does not persist beyond a few months. But these studies stand in stark contrast to the experience of many patients and, indeed, as we shall see, many practitioners of ECT. The weaknesses of the studies and the failure of psychiatry to openly communicate all that it knows – and does not know - about the memory effects of ECT provide the most dramatic illustration of psychiatry's "Don't Look, Don't Tell" policy regarding ECT. This policy expresses itself in several interrelated areas:

1. The gap between psychiatrists' public statements and private experience regarding ECT and memory loss.
2. The methods by which memory loss is studied – or not studied.
3. How study results are published – or not published.

The 1990 Task Force Report – Hear No Evil, Speak No Evil

The 1990 APA task force report on ECT devoted 2 pages (out of 124) to the topic of "Cognitive Side Effects" and only a few sentences to memory loss. The report acknowledged the "loss of specific memories for some events that occur over the months immediately preceding, during; and following the treatment course (Squire 1986)" but stated that "objective testing does not indicate that capacities …to remember information from the past are persistently impaired by ECT (Squire 1986; Taylor et al. 1982; Weeks et al. 1980) A small minority of patients, however, report persistent deficits (Freeman and Kendell 1986). The basis of these complaints is not well understood."[70]

The APA's short summation of memory loss in 1990 was false on two counts and there is every reason to believe they knew it.

First, the sole study cited for the claim that a "small minority" of patients report persistent deficits is a 1986 Freeman and Kendell study in which patients were interviewed by psychiatrists

in the hospital where they had received ECT. The authors admitted that this means of acquiring data about patient attitudes was open to criticism:

> It is obviously going to be difficult to come back to a hospital where you have been treated and criticize the treatment that you were given in a face-to-face meeting with a doctor…. It was our impression that those patients who had strong views spoke out with little inhibition. What is less certain is whether there was a significant number of people in the midground who felt more upset by ECT than they were prepared to tell us.[71]

One could also argue that those who were most impaired by the treatment would be among the least likely to be interviewed. For example, out of a potential pool of 243 interviewees for this study, 3 refused to be interviewed, 4 had committed suicide, and the deaths of two others "may have been related to ECT."[72] Nevertheless, the study found that "30% [of ECT patients] agreed with the statement that their memory had never returned to normal afterwards…. Twenty-eight percent felt that ECT caused permanent change to memory…."[73] The authors wrote, "We were surprised by the large number who complained of memory impairment. Many of them did so spontaneously without being prompted, and a striking 30% felt that their memory had been permanently affected."[74]

Thirty percent is hardly a "small minority," particularly considering the circumstances of the interviewing.   Several other studies known to the task force commented on the large number of patients who reported persistent memory loss.   These studies were cited in the task force report, but their conclusions regarding patient memory loss complaints went unmentioned. The Weeks study asked the question, "If no permanent deficit in memory is caused by ECT, why do so many patients complain of both temporary and lasting memory impairment?"[75] The 1986 Squire study found that, "Many patients who have received ECT continue to report even several months after treatment that their memory is not as good as it used to be, and they attribute their memory problem to the ECT experience."[76]

Squire was the leading ECT memory loss investigator of the 70's and 80's. He authored numerous studies on ECT and memory loss. A 1979 Squire study was also cited by the task force report (p. 109) – but not in the section on cognitive side effects. In that study Squire, referencing two earlier studies, wrote, "We recently reported that memory complaints were common 6 to 9 months after a course of bilateral ECT, being reported by 60% to 70% of patients interviewed (Squire and Chace, 1975; Squire, 1977)"[77] The 1975 Squire and Chace study referred to here found that, "Complaints about memory function six to nine months after ECT were not equally distributed among the three follow-up groups…. The percentage of subjects with memory complaints was 63% after bilateral ECT, 30% after unilateral ECT, and 17% after hospitalization without ECT."[78] A 1983 study by Squire and Slater found, "Fifty-five percent felt that their memories were not as good as those of other people of the same age and that this was related to their having received ECT."[79]

In short, the APA task force had to ignore the conclusions of at least seven studies (Squire 1986, Freeman and Kendall 1986, Weeks et al. 1980, Squire 1979, Squire and Chace 1975, Squire 1977, Squire 1983), including three that they cited, in order to claim that only a small

minority of patients reported persistent deficits. No study was cited that actually backed up the "small minority" claim.

(A 2003 systematic review of studies that investigated patients' views of ECT found, "The rate of reported persistent memory loss varied between 29% and 55%."[80] The review, published in the *British Medical Journal*, cited one study in which a third of those who had received ECT agreed that "electroconvulsive therapy permanently wipes out large parts of memory."[81] The 2003 study also found, not surprisingly, that when patients are interviewed by the staff of the hospital where they received ECT, their responses tend to be more favorable to ECT.)

The task force also misrepresented the data - and again misreported the conclusions of the very studies it cited - when it claimed that "objective testing" did not support the idea that ECT produced persistent memory impairment. The 1986 Squire study found "a persisting impairment [in autobiographical memory] was present …."[82] In this study individuals who had received bilateral ECT were asked 10 questions about their personal history. When the pre-ECT and 7 months post-ECT answers to all 10 questions were combined together, statistical analysis indicated that ECT appeared to have produced no memory deficit.

But when the authors examined the answers to three questions regarding events that had occurred anywhere from 6 to 37 months pre-ECT, there was clear evidence of persisting memory deficits. Squire wrote,

> Control subjects recalled an average of about 13 details concerning these three events at the time of the pre-ECT test and recalled almost as much information 7 months later, without intervening ECT. The ECT patients initially recalled a little more information than the control patients about these same three events (about 16 details per person). However, 7 months later they forgot much of what they had previously reported and now recalled only 7 details per person."[83]

Squire also said,

> It is not yet clear how to evaluate the finding that at seven months after treatment persons who had received bilateral ECT occasionally failed to recognize as familiar even remote events that had occurred many years ago. Specifically, 5 of the 10 persons in our sample denied familiarity to a total of 18 remote events that they had reported as facts before ECT, seven months earlier.[84]

Even more revealing is the fact that the task force's discussion of cognitive side effects failed to even mention two important papers by the chairperson of the 1990 task force report himself, Richard Weiner.

In 1984, Weiner published a 53-page paper in *The Behavioral and Brain Sciences* entitled, "Does electroconvulsive therapy cause brain damage?"

In this paper Weiner was critical of the 1980 Weeks study that was one of three studies cited by the 1990 task force as evidence that memory was not persistently impaired by ECT. Weeks

gave a battery of tests to 51 subjects who received ECT (15 unilateral, 36 bilateral) and 51 patients matched to the ECT group on age, sex, social class, educational attainment, and severity of depression. According to Weiner,

> Unfortunately, their personal memory testing paradigm, which was described only as 'an interview schedule with 28 items sampling memories from various times in the subject's life, from early childhood to the present' (p. 36), also demonstrated no intergroup [ECT vs. non-ECT] differences for the acute post-ECT test session. This raises some concern regarding the questionnaire's overall sensitivity.[85]

"Some concern" is putting it mildly. By 1990 it was well known that acute post-ECT patients demonstrate significant memory loss. Squire noted significant autobiographical memory impairment within one week post-ECT in a 1981 study.[86] The 1990 APA report itself mentions "retrograde memory disturbance during the treatment course" and states, "The available evidence indicates that these side effects subside during the weeks following the treatment course."[87] Whether the trouble lay in the questionnaire or some other factor (e.g., combining the test results of the bilateral and unilateral groups), Week's study was clearly faulty; but the APA nevertheless offered it into evidence.

In 1986 Weiner had published another important memory study that concluded, "The above results represent provocative evidence for what amounts to objective personal memory losses lasting at least six months with BL but not with UL ECT, and represents the first time such a differential effect has been reported."[88] In this study, the pulse bilateral group failed to recall 30% of the items they had recalled before ECT. The 30% statistic was a mean. Thus, some subjects "lost" over 30% of their memories. How high that percentage went in some subjects was not revealed in the study. Weiner wrote that his findings suggested the cause of this memory loss was "organic rather than functional."[89] In other words, the memory loss was not simply the result of depressed mood. This conclusion, Weiner stated, was "supported by a number of highly significant correlations between acute objective memory test changes and acute EEG abnormalities."[90]

None of this information made it into the 1990 task force report. One is forced to conclude that the 1990 APA task force report's discussion of ECT and memory loss ignored critical evidence related to ECT's memory effects. Politics and PR won out over medical science.

The 2001 Task Force Report

Eleven years after the 1990 task force report the APA produced a second report on ECT which, for the first time, admitted that permanent memory loss can occur. It stated,

> A few days after the ECT course, memory for events in the remote past is *usually* intact, but there may be difficulty in recalling events that transpired several months to years prior to ECT. The retrograde amnesia over this time span is *rarely* complete. Rather, patients have gaps or spottiness in their memories of personal and public events.
>
> In some patients the recovery from retrograde amnesia will be incomplete, and evidence has shown that *ECT can result in persistent or permanent memory loss*.[91] (emphasis added)

21

This confession did not come without opposition. Harold Sackeim, one of the world's leading experts on ECT and a member of both the 1990 and 2001 APA task forces, discussed the contention surrounding the issue of memory loss in a March 14, 2004 deposition (the case was Akkerman v. Johnson, Court of the State of California for the County of Santa Barbara, Anacapa Division, Case No. 01069713.) Sackeim's answers to the deposition questions were most revealing.

> Q: Today you think doctors should tell patients they could experience severe long-term memory loss?
> A: Um hm.
> Q: Yes?
> A: Yes.
> Q: Do you think they should have told them that three years ago?
> A: And this is what I will say again. I don't think three, four years ago people would have been in a position to know that that was either an outcome in their patients, something that would be expected as part of community standards, because it wasn't.[92]

> Q: Now, what changed in the last three years that leads you to believe that psychiatrists wouldn't have known, say, in 1999 -- let's make it 1999 -- what's changed since 1999 to make it that now psychiatrists should know that ECT causes severe memory loss that they didn't know then?
> A. There are a couple of things that are of consequence. The field itself never really had an opportunity to have a discussion about patients who have complaints about long-term memory loss, who, for instance, believe that the treatment saved their lives, there's no issue about their motivation for making those types of complaints.[93]

Sackeim then expanded upon his answer:

> A: There was an opportunity, almost a watershed moment for the field in California when a couple hundred practitioners in a couple-day course on ECT -- I was lecturing on the issue of cognition and lecturing about long-term consequences. And one of the very famous people in the field [Richard Abrams? See concluding remarks below.] got up, said this doesn't happen, just doesn't happen.
>
> And there was confrontation where I asked the audience, had they seen it, do they have patients who they believe generally have long-term negative effects. And two-thirds raised their hands. And some other very well known people in the field said, yes, that happens.
>
> And so that was the first time publicly that the field itself said no to the position that it can't happen. And that was then taken up in the APA task force report, which was being written at the time. It was discussed at length because it was a very -- it was a new and different position for the American Psychiatric Association to take and very controversial, with again, some of the -- like there's a left wing, there's a right wing, opposing, this type of perspective.
>
> And in the context of this, the NIMH funded the largest prospective study of ECT in the community, ever, of how ECT is practiced in the community and in terms of its cognitive effects. One of the limitations in being able to discuss the cognitive effects is all of the studies long-term are small, small samples. You don't see much. I mean, particularly, if there are going to be some people who have outliers. So that study has revealed effects at six months.

So are you asking, is there different information? Yes, there is. Is there a different attitude in the community? Yes, there is.[94]

Sackeim's testimony illustrates how psychiatrists have historically approached the question of memory loss and ECT. Sackeim is discussing a conference that occurred at the same time that the APA's 2001 task force report on memory loss was being written, around 1999 or 2000. According to Sackeim's testimony this conference was the first time that psychiatrists "had an opportunity" to have a discussion about patients who have complaints about long-term memory loss.

Since psychiatrists had been using ECT for 60 years at this time one has to wonder why psychiatrists never found a time to discuss memory loss, particularly since 1) two-thirds of the psychiatrists attending this conference had seen "long-term negative effects" in their patients; and 2) decades of evidence showed large numbers of patients complaining about memory loss. Earlier practitioners of ECT surely saw long-term effects every bit as negative as those witnessed by Sackeim's audience.

Nevertheless, psychiatrists, in Sackeim's view, were, in 1999, simply "people" who were not "in a position to know" that severe long-term memory loss could be an outcome for their patients until an "opportunity" presented itself in a conference held 60 years after psychiatrists began using ECT.

Sackeim's testimony suggests that for 60 years psychiatrists were aware that shock caused significant memory loss but avoided discussing it, either amongst themselves or "publicly." For 60 years the no-memory-loss hard-liners were allowed to rule, even though the majority of ECT practitioners had witnessed significant memory loss in some of their patients.

Practicing not-knowing

60 years of silence must somehow be justified. One way is to question the motives of patients who claim to have suffered memory loss, as Sackeim does. Unless the patient felt overwhelmingly positive ("saved their lives") about ECT, they were not to be trusted.

Another means of justification is to simply choose to not know about memory loss – to make little or no attempt to find out what happens to individuals who undergo ECT. There are at least two methods to do this. 1. Don't follow up with patients to determine the outcomes of treatment. 2. Don't do the studies necessary to determine the duration and extent of memory loss. These methods of justification are, of course, interrelated.

The first means of not knowing is commonplace in psychiatry.   In 1995 the *Journal of Neuropsychiatry and Clinical Neurosciences* published the results of a survey of members of the American Neuropsychiatric Association and the British Neuropsychiatry Association "to determine the extent to which neuropsychiatrists employ formal measures of clinical outcome." Survey results revealed that, "formal diagnostic evaluations and outcome measures were rarely applied consistently to the broad range of neuropsychiatric conditions encountered clinically."[95] According to the report, "… the findings suggest that practicing clinicians are currently unable to

contribute tangibly to the information base regarding efficacy of treatments used in neuropsychiatry."[96]

There is no obvious reason why these survey results should not apply specifically to neuropsychiatrists who practice ECT. That they do is suggested by an editorial written by Edward Coffey (lead author of the 1995 report referenced above and a member of the 2001 APA task force) and published in *The Journal of ECT* in 2003. Coffey wrote, "…we lack consistent data on the variation in ECT safety outcomes in this country, including our patients' perceptions of the safety of their ECT care. Of course, it is precisely these specific data that are of greatest interest to our patients." He added, "…our field lacks data on the rates of adherence to ECT and the factors associated with it" and he "challenge[d] all ECT practitioners to begin systematically collecting outcome data for their individual practices."[97]

The second means of not knowing about memory loss and ECT is not to study it. In view of the number of ECT studies which have focused on memory deficits, it might appear that psychiatry cannot be charged with failure to investigate. But, as we shall see, the charge is not without grounds.

We previously mentioned a 1984 paper by Richard Weiner, who was the chairperson of both the 1990 and 2001 APA task force reports on ECT. In that paper Weiner reached the following conclusions regarding ECT and memory loss:

> Accordingly, it is still unclear to what extent self-rated amnestic deficits are present long after ECT. Furthermore, it is also uncertain whether such findings, if present, are based upon a true organic deficit not detectable by standard objective measures of memory testing, or whether they represent some other, more functionally related, etiology."[98]

> In summary, the results of numerous studies of memory performance after ECT still have not provided a definitive answer to the question of whether and if so, to what degree ECT is associated with persistent memory loss. … reliance upon 'mean' scores may well overlook the possibility that if substantial long-term deficits occur with ECT, they do so rarely. … the recent Royal College of Psychiatrists survey… showed that British psychiatrists estimated a 1% incidence for persistent deficits with unilateral ECT and 2% with bilateral ECT, and that general practitioners responded to a somewhat differently worded query with a 7% incidence for ECT in general. *The only systematic way to consider such a low-incidence phenomenon is to study large numbers of ECT and control subjects with sufficiently sensitive measures and to focus on individual responses in addition to group means.* (emphasis added).[99]

In view of many of the points raised earlier in this paper, the value of surveys of psychiatrists in determining the prevalence of permanent memory loss as a result of ECT is highly questionable – certainly not adequate to regard memory loss as a "low-incidence" phenomenon.

But Weiner's ultimate conclusion was valid, if rather limited. There was a need to study large numbers of subjects and focus on individual responses in addition to group means. Reporting only the mean autobiographical memory deficits for a large group can hide the memory loss in a significant portion of the group.

It also seems obvious that one would search for persistent memory loss where it is most likely to occur.   It was known at that time, and became even more apparent later, that bilateral ECT and high-dose (2.5 times threshold) conditions of ECT are both associated with an increase in the severity of cognitive side effects.[100] Surely if the intent is to determine the degree to which ECT can cause persistent memory loss, research should focus on the type of ECT most likely to produce such loss – high-dose, bilateral ECT.

Finally there was a need to study memory effects at least two months after ECT to assess whether the memory loss was persisting. (In the Sackeim deposition referenced above, Sackeim testified as follows regarding memory loss: "My view is, if it's there at two months, it's probably going to be there forever."[101])

In short, to truly sort out the memory loss question, it should have been clear in 1984 that there was a need to study large numbers of patients who received high-dose bilateral ECT - with a focus on individual responses - at least two months post-ECT.

This has never been done.

In looking through the 1990 and 2001 APA task force reports and using other sources I compiled the following list of post-1984 published, peer-reviewed studies (or articles) that examined autobiographical memory loss at least two months post-ECT.

1. Weiner et al., "Effects of Stimulus Parameters on Cognitive Side Effects," *Ann N Y Acad Sci*, 462:315-325, 1986.
2. Sackeim et al.(a), "Cognitive consequences of low-dosage electroconvulsive therapy," *Ann N Y Acad Sci*, 462:326-340, 1986.
3. Squire LR, "Memory functions as affected by electroconvulsive therapy," *Ann N Y Acad Sci* 462:307-314, 1986.
4. Frith et al, "A comparison of some retrograde and anterograde effects of electroconvulsive shock in patients with severe depression," Br. J Psychol. 1987;78:53-63.
5. Squire, Zouzounis, "Self-ratings of memory dysfunction: different findings in depression and amnesia," J Clin Exp Neuropsychol 10:727-738, 1988.
6. Tulving E et al., "Priming of semantic autobiographical knowledge: a case study of retrograde amnesia. *Brain Cogn.* 1988;8:3-20.
7. Calev et al., "Early and long-term effects of electroconvulsive therapy and depression on memory and other cognitive functions," *Journal of Nervous and Mental Disease*, Vol 179(9), 526-533, Sept 1991.
8. Sackeim et al., "Effects of Stimulus Intensity and Electrode Placement on the Efficacy and Cognitive Effects of Electroconvulsive Therapy," *The New England Journal of Medicine*, 328:839-846, 1993.
9. Sobin, Sackeim, et al., "Predictors of Retrograde Amnesia Following ECT," *Am J Psychiatry*, 152:7, 995-1001, July 1995.
10. McElhiney et al., "Autobiographical memory and mood: effects of electroconvulsive therapy," *Neuropsychology* 9:501-517, 1995.
11. Coleman, Sackeim et al., "Subjective Memory Complaints Prior to and Following Electroconvulsive Therapy," *Biological Psychiatry*, 39:346-356, 1996.
12. Sackeim et al., "A Prospective, Randomized, Double-bind Comparison of Bilateral and Right Unilateral Electroconvulsive Therapy at Different Stimulus Intensities," *Arch Gen Psychiatry*, Vol 57, 425-434, May 2000.
13. Lisanby et al., "The effects of electroconvulsive therapy on memory of autobiographical and public events," *Arch Gen Psychiatry* 57:581-590, 2000.

An analysis of these studies tells much about what those who study ECT are willing and not willing to investigate and reveal.

The Post-1984 Autobiographical Memory Studies

Several of the studies listed above can be excluded from consideration here.

Study #2 was a "low-dose" study.

Study #3 was not a new study, just a review of Squire's earlier studies, which, as Weiner said, did not provide a definitive answer. Squire briefly discusses one anterograde memory study from 1985, and he cites Weiner and Sackeim's 1986 studies (#1 and #2 above), although he neglects to mention Weiner's findings of persisting autobiographical memory loss. All of the other studies Squire cites are from 1984 or earlier. He does, however, refer to a 1981 study in which he found "… at seven months after treatment persons who had received bilateral ECT occasionally failed to recognize as familiar even remote events that had occurred many years ago."[102]

Study #4 is, essentially, part 2 of the Johnstone sham ECT study (Johnstone et al., "The Northwick Park electroconvulsive therapy trial," *The Lancet*, 20-27 December, 1317-1320.) 62 patients completed the full course of treatment – 2 bilateral shocks per week for four weeks. This is significant in itself since shock in the U.S. is given three times a week, which has been associated with more cognitive deficits. Study #4 states, "… the no shock group had caught up with the shock group within one month after the end of treatment."[103] In other words, the sham-ECT and real-ECT groups were equivalent in depression symptoms after 1 month (about the time the EEG effects have worn off). This study involved two remote memory tests: 1) recognition of famous names from the past; and 2) sentence verification in which, "A subject was presented with a list of sentences. Half of these made sense (e.g., oranges can be bought in shops) and half did not (e.g., oranges move around searching for food). The subject had to indicate whether each sentence was true or false by making a cross or tick beside it."[104] It can be seen that neither of these tests was a good measure of autobiographical memory impairment.

Study #5 was not long-term and involved self-ratings, not objective ratings, of memory.

Study #6 was a case study.

Study #7, by Avraham Calev, has so many problems that one hesitates to even mention it. Calev wrote, "... due to circumstantial problems, such as absence of the tester, *noncooperation or refusal on certain tasks*, or absence of relatives to corroborate personal memory items, not all patients were available as subjects for all tests.... Another reason ... was that *not all patients had reached 6-month follow-up by the time the present analysis was conducted.* Nevertheless, at least 10 subjects [out of 27 = 37%] were available for each test on all occasions, including 6-month follow-up [emphasis added]."[105]

Clearly a 37% follow-up rate, non-cooperation and refusal are poor indicators. (Contrast this with Sackeim et al. 1993: 66 of 96 (69%) of patients tested at two-months, Sackeim et al. 2000: 55 of 80 (69%) tested at two months; and Weiner et al. 1986: 39 of 53 (74%) tested at 6 months). This suggests significant selection bias may have been at work. The most seriously impaired subjects may not have participated in the six month follow-up. In addition, 6 of Calev's subjects received relatively few treatments, between 4 and 7, and *all patients* were dosed at "150% of initial threshold," not the 2.5 times threshold that generally defines a high-dose condition.[106]

Calev's report that, "There was no change in depressive symptoms [among his patients] from 1- to 6-month follow-up,"[107] casts further doubt on his results. Worsening depression at six months is a common occurrence.

Moreover, Calev found no temporal gradient in the memory loss of his patients.[108] Calev himself wrote that retrograde memory deficits due to ECT are "reportedly characterized by an amnestic time gradient, whereby the distant past is remembered better than more recent events. (e.g., Calev et al. 1989, Squire et al., 1981). Events related to the period immediately prior to ECT administration are reported to be least well remembered or permanently lost (Squire et al., 1981)."[109] It is an accepted fact of ECT practice that patients will lose memory of the period surrounding the treatment. Lisanby's conclusion in her 2000 study is typical: "In line with traditional views [3 studies cited], this study supported the notion that a temporal gradient characterizes the memory deficits after ECT….." [110] The 2001 APA task force report states, "Deficits in recalling both personal (autobiographic) and public information …are typically greatest for events that occurred temporally closest to the treatment."[111] The fact that Calev found no such gradient further increases our suspicion that there were serious flaws in his study.

Calev used two tests to examine retrograde memory effects. The Famous Events Questionnaire testing showed no effect of ECT at 6-months. The overall results were illustrated in a graph.

Calev's other retrograde test, which focused more directly on autobiographical memory, was the Personal Memory Questionnaire. Calev did not display the results of this questionnaire in any table or graph, saying only that "patients were more impaired in recall of personal events at the post-ECT testing … than they were at the 6-month follow-up …."[112]

We are left with Weiner's 1986 study (#1) and the last 6 studies. 53 subjects received ECT in Weiner's study. Study #8 involved 96 patients, study #9 involved 71 patients, study #10 involved 75 patients, study #11 involved 70 patients, study #12 involved 80 patients and study #13 involved 55 patients.

It would appear that 500 subjects received ECT in these 7 studies. However, the 70 patients in the Coleman study were part of the 96 patients in the 1993 Sackeim (b) study.[113] Also, McElhiney refers to the 75 patients in his study as a "subsample" of a "larger parent study," also the 1993 Sackeim study.[114] Finally, Richard Abrams, in a 2002 editorial in *The Journal of ECT*, revealed that the Lisanby study "analyzed a differently focused subset of autobiographical data

collected during the course of" Sackeim's 2000 study (Sackeim et al. (c) above).[115] Thus we end up with 300 unique patients in 4 studies who received ECT.

Looking at these studies more closely, we find that in all except the Weiner study patients were randomized to one of four treatment groups, low and high-dose unilateral (UL) ECT, and low and high-dose bilateral (BL). Weiner divided his patients into pulse and sine wave unilateral and pulse and sine wave bilateral. Thus, not all subjects in these studies received high-dose bilateral ECT. Approximately 182 of these patients received high-dose, bilateral (HDBL) ECT, either as part of an initial course or as crossover treatment. (See table below.) Approximately 132 of these HDBL patients were tested at 2-6 months.

The actual test results, however, were not always published.

Weiner did publish his "long-term [6 months] personal memory impairment" results as a bar graph in Figure 2 of his study. The graph shows that pulse and sine-wave unilateral patients suffered the least impairment - an approximate 17% - 18% mean drop in items recalled. The pulse bilateral group suffered a 30% drop in the number of autobiographical memory items recalled, with the sine wave bilateral group demonstrating a nearly 40% drop.

Sackeim (#8) published the short-term (one week post-ECT) results of his retrograde autobiographical memory interviews, but no long-term results. The short-term recall consistencies (defined as the percent of responses that matched their pre-ECT responses) for the high dose bilateral group were 76%±13.[116] However, McElhiney (#10), whose study involved a "subsample" of Sackeim's study, did publish the long term autobiographical memory testing results for 25 Sackeim patients who received BL ECT. (It is unclear exactly how many of the 25 received some high-dose BL ECT. Presumably most did.) The mean consistencies (liberally defined as the percent of responses at 2 months post-ECT that matched *either* their pre-ECT responses or their 1 week post-ECT responses) were around 50%.[117]

Sobin (#9) also did not publish any two-month autobiographical memory results. However, she did note the existence of long-term deficits: "Pre-ECT global cognitive status and the duration of postictal disorientation were strong predictors of the magnitude of retrograde amnesia in the week after the course *and at 2-month follow-up*."[118] (emphasis added)

As with his 1993 study, Sackeim's 2000 study (#12) revealed only the short-term results of his autobiographical memory interviews. Inconsistencies (defined as "percentage of factual responses inconsistent with baseline") for the high-dose bilateral group were -40.4±10.0.[119] It might be hoped that Lisanby could shed some light on the long-term autobiographical results, since she analyzed a subset of Sackeim's autobiographical data. Unfortunately, Lisanby's bar graphs show 2-month results for *all patients* (*mean scores for unilateral and bilateral combined*). The autobiographical memory results for the estimated 28 high-dose BL ECT patients are not revealed.

Thus we have actual published autobiographical memory data for only 45 unique individuals who received high dose bilateral shock: the 20 in Weiner's 1986 study and the 25 in Sackeim's

1993 study, whose memory outcomes were revealed by McElhiney (see table below). In none of these studies is there the "focus on individual responses in addition to group means" that Weiner requested. However, all of the *published* autobiographical memory loss results at two months demonstrate considerable memory loss in ECT subjects.

The results of our examination of the memory loss studies are summarized in the following table:

Study	No. Patients who received high dose (HD) BL shock[120]	No. HDBL patients tested at 2 months[121]	Long term autobiographical memory deficits for HDBL subjects published?
Weiner	29	20	Yes – 20 patients
Sackeim 1993	27 + 34 crossover	42	Yes (in McElhiney 1995) - 25 patients
Sobin	18 + 17 crossover	35	No
Sackeim 2000	20 + 36 crossover	35 (maximum, may be less)	No
Total	182	132	45 patients

All of these studies had significant drop-out rates. Many patients did not show up for the long term follow-ups, so we have to wonder if there is a selection process going on in which the results for subjects with the worst outcomes are not reflected in long term statistics. Sackeim commented on this in his 2000 study: "Only 55 patients (69%) were evaluated at the 2-month follow-up. This raises the possibility of selection bias caused by selective loss to follow-up of patients with more pronounced cognitive deficits."[122]

Finally, Weiner's call for a "focus on individual responses in addition to group means" continues to go unanswered.

Clearly psychiatry's examination of the memory loss question has been anything but definitive. Long-term memory loss data for high-dose bilateral patients is seldom published, although when it is, it indicates massive memory loss at 2-months.

As dismal as these results are, however, the memory loss produced by ECT as actually practiced in the community may be even worse. The reason for this involves a little known fact of ECT practice that we will discuss next.

Ultrathreshold ECT

The memory studies that we have so far examined were, of course, all conducted according to particular research parameters. One of those parameters is known as the seizure threshold, which is generally defined for each person as the level of electrical charge necessary to produce a seizure lasting at least 25 seconds. The subject's seizure threshold is determined by titrating (gradually increasing) the initial dose of electrical charge until a seizure lasting at least 25 seconds is produced. High dose subjects are then treated at 2.5 times their initial seizure

threshold. For example, it may initially take 60 millicoulombs* to produce an adequate seizure in a subject. If that person is then assigned to the high dose condition, subsequent shocks would be delivered at 2.5 x 60, or 150 millicoulombs (or higher, see below).

When researchers examine the memory effects of ECT, the treatments are typically administered using titration. Current is delivered at no more than 2.5 times the seizure threshold. In all of the more recent memory studies we examined above (#7-#13) titration was employed. This practice is observed because, "… the degree to which ECT stimulus intensity exceeds the seizure threshold may be an important determinant of both therapeutic effectiveness and cognitive side effects."[123] Put another way, "Once above the threshold, higher intensities of electrical stimulation may result in greater cognitive disturbance."[124]

(As we mention previously, one study of unilateral ECT found the relationship between stimulus intensity and cognitive side effects to extend to doses as high as 12 times threshold. In that study the authors found, "the likelihood of both antidepressant response and cognitive deficits increased as stimulus dose increased relative to initial seizure threshold, up through 8 to 12 times the threshold."[125])

For this reason, the 2001 APA task force recommends, "Patients treated with bilateral ECT generally should receive moderately suprathreshold stimulation, defined as between 50% and 150% above seizure threshold (1.5-2.5 times threshold)."[126]

Since patients in these research studies are treated at no more than 2.5 times their seizure thresholds, the research results would therefore not apply to individuals treated at more than 2.5 times their seizure threshold.

But it is routine practice among psychiatrists who deliver ECT to administer doses that are much higher than 2.5 times threshold. According to Harold Sackeim, ECT is sometimes practiced "using electrical intensities far in excess of that needed to produce seizures," a practice that "undoubtedly contributes to adverse cognitive side effects."[127]

Seizure thresholds may vary by 4000%. Sackeim writes,

> In our clinical practice, the range of minimal dose needed to produce seizures is even greater, on the order of 40-fold.[128]

> Even with an efficient electrical waveform, however, the true variability in what is minimally needed to produce a seizure may be 40-fold. Consequently, some patients may receive at each and every treatment an electrical dose that is grossly in excess of their threshold (e.g., by up to 4000%).[129]

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* 1 coulomb is the amount of electrical charge carried by a current of one ampere for one second. It equals 6.242×10^{18} electrons. A millicoulomb is one thousandth of a coulomb.

He then compares this to drug dosing:

> The typical therapeutic dose of imipramine is approximately 150 to 350 mg/day. Were we to treat patients at 10 to 20 times this range, let alone 40 times the typical therapeutic dose, there would be marked toxicity and, in many cases, death. Fortunately, with respect to *medical complications and mortality*, ECT has a considerably wider margin of safety. However, the practice of using electrical intensities far in excess of that needed to produce seizures undoubtedly contributes to adverse cognitive side effects."[130]

Despite this wide variability in seizure threshold, many ECT providers do not titrate. A 1992 survey found that "only 39% [of ECT practitioners] routinely measured seizure threshold."[131] Also, according to Sackeim, "Commonly, clinicians do not know the degree to which they are exceeding seizure threshold when setting electrical intensity. For some patients with low thresholds, even relatively moderate settings on the device will produce a stimulus that may exceed threshold by 500% or 1000%."[132]

Psychiatrists who do not titrate use certain formulas to select an initial ECT dose. These formulas are not accurate. Many factors influence seizure threshold, including electrode placement, gender, and age. According to Sackeim, "a substantial proportion of the variability in threshold is due to *individual differences* in degree of current shunting."[133] In another study Sackeim found that only 47.1% of the variance in seizure threshold could be accounted for using sex, age, and treatment modality (UL or BL) to predict threshold. He concluded, "This analysis also indicated that, while the predictors each made important independent contributions, the majority of the variance in seizure threshold was still unaccounted for."[134] Weiner also studied thresholds and found, "these variables [electrode placement, gender, and age] accounted for 50% of the variance in seizure threshold…."[135] According to the APA's 2001 task force report, "In general, the most complex formulas account for only 40% or less of the variability in initial seizure threshold."[136] (The task force report cites 7 studies for this statistic.) The task force also states, "Because of limited success in predicting the wide individual differences in seizure threshold on the basis of patient or treatment factors, empirical titration provides the most precise method for quantifying seizure threshold."[137]

Thus, without titration, any estimate of threshold must be highly suspect. And if only 39% of ECT practitioners are titrating, there can be no doubt that a significant number of subjects are shocked at doses much higher than 2.5 times their thresholds.

Rasumussen pointed to this danger in a 2002 commentary in *The Journal of ECT*. Responding to a previous article by Richard Abrams, Rasumussen wrote,

> Abrams cites the fact that, of numerous titration studies he reviewed, approximately two-thirds of thresholds were in a threefold range. Thus, approximately one-third of thresholds lie outside the threefold range. For such patients, the fixed or age-based does may well be outside of an acceptable range of threshold multiples. *On a busy ECT service, this may amount to over 100 per year potentially treated with an inadvisable electrical dose.*[138] (emphasis added)

Another factor that may be of significance is the tendency of thresholds to increase during a course of ECT. Psychiatrists typically compensate for this tendency by automatically increasing the stimulus level during the course of treatment.

However, not all individuals display an increased seizure threshold after several treatments. Coffey and Weiner did a study in which they titrated before the first and again before the sixth treatment. They found that only 60% of men aged 40 to 49 experienced such an increase[139] and of the four males who received BL shock, only 1 (25%) showed an increase in threshold prior to the sixth treatment.[140] According to Michael Alan Taylor, "Seizure thresholds increase over the course of treatment in about 30%-40% of patients, so energy doses may need to be increased after three to four treatments."[141] Automatically increasing stimulus doses may also contribute to shocking patients at ultrathreshold levels.

All of this bears upon the question of memory loss. Psychiatrists have never studied the effects of bilateral ECT when administered at doses over 3-times threshold. Yet, as we have shown, ECT at such levels is not uncommon. The effects of this practice, including memory effects, have never been studied and are unknown, at least as far as the published literature is concerned. Thus the current body of ECT research, which is completely inadequate for drawing conclusions regarding the memory effects of ECT at sub 3-times threshold levels, is even more useless for drawing conclusions about the memory effects of ECT as currently practiced by the majority of shock practitioners. However, if the relationship between stimulus dose and cognitive deficits demonstrated by unilateral ECT (up to 12 times the threshold) is any indication, the ultrathreshold effects of bilateral ECT cannot be good.

Sackeim's analogy to drug testing is fitting. We cannot draw conclusions about the effects of a drug administered at high doses by only studying the effect of the drug administered at low doses. Yet this is precisely what psychiatrists and ECT researchers have done.

ECT and Brain Damage – the MRI studies

In 1991 Edward Coffey published the second of his two major MRI studies of ECT patients – a study of 35 patients who were given BL ECT.[142] In the introduction to this study Coffey took care to point out the shortcomings of previous research and wrote that before his 1991 study, only four prospective studies had been reported and only two of them used MRI. One of the previous MRI studies was Coffey's 1988 study. The other was Pande's study, which involved only 7 patients and MRI was done only 1 week post-ECT.

We will focus on Coffey's 1991 study, since Coffey himself acknowledges that previous studies had not answered the question of ECT and the type of brain damage that might show up on an MRI.

There are several problems with Coffey's 1991 study. They generally concern establishing how intense the shocks were. To determine whether shock could produce damage visible on

MRI, it would surely be prudent to study the effects of high intensity shock and high numbers of treatments on a significant number of subjects. Coffey, predictably, did not do this.

The mean number of treatments was 9.6±3.0.[143] Therefore, a number of these patients received less than 9 shocks. "The total number of ECT treatments was determined by each patient's physician and was based on therapeutic response."[144]

"Six patients did not receive the third [MRI] image; one died of unknown causes in a nursing home, one became pregnant during the follow-up interval, and *four refused further involvement with the study*."[145] (emphasis added) So only 29 subjects were tested at 6 months, with possibly the worst results selected out.

All we know about the stimulus dose was that "stimulus dosage variables were adjusted to elicit seizures of at least 25 seconds by standard electroencephalographic criteria."[146] We do not know how many of these subjects were shocked at 2.5 times threshold.

The study presents no overall information on response, relapse, or post-ictal disorientation, indicators that might indirectly give a clue as to the intensity of the shocks, since the degree to which the dose exceeds threshold is related to therapeutic outcome as well as cognitive effects.

We do know, "Fifteen patients had received one or more previous courses of ECT,"[147] and, "Many of the patients exhibited structural abnormalities on their baseline (pre-ECT) brain MR images."[148] Several had cortical atrophy, lateral ventricular enlargement and various hyperintensities. Thus, Coffey was not dealing with ECT-naïve patients, but patients who already exhibited abnormalities to which ECT may have contributed. The degree to which previous damage might limit the brain's response to subsequent ECT is not discussed by Coffey.

Despite all this, "In five subjects, the pairwise global comparisons revealed an apparent increase in subcortical hyperintensity, most likely secondary to progression of ongoing cerebrovascular disease during follow-up."[149]

Most likely? That's hardly conclusive. The authors say, "Although we failed to find evidence of changes in brain volume after ECT, this does not mean that such changes did not or could not have occurred, since even modern brain imaging technologies have certain inherent limitations."[150]

Coffey also admits, "… it is possible that changes in neuronal density could have occurred that were not detectable with the methods available in this study." He confesses, "… it is possible that subtle atrophic effects in a particular structure may be obscured when that structure is combined with other adjacent structures in the same region."[151]

Finally he adds, "Still, it is difficult to interpret with certainty worsening of brain MR imaging abnormalities after ECT in the absence of control data describing the natural course of such brain abnormalities. Unfortunately, few data are available on the natural course of subcortical hyperintensity."[152]

We don't know how many of the 26 subjects in this study who were tested at 6 months received at least 9 shocks at 2.5 times threshold. Less than 20? Less than 10? This study cannot be considered to even approach being conclusive. Indeed, three years after his 1991 study Coffey himself wrote, "Clearly, prospective studies that compare pre- and post-ECT imaging data are required to determine whether ECT causes changes in brain structure."[153]

And were those "required" studies ever done? No.

A Medline search under "Electroconvulsive and MRI" failed to uncover a single post-Coffey (1991) prospective MRI study of ECT's effects on brain structure.

Nevertheless, other ECT researchers are happy to write about this issue as if it has been completely resolved. In July, 1994, we find Devanand et al. claiming, "There is no credible evidence that ECT causes structural brain damage,"[154] even though,

1) He admits "there is a paucity of quantitative data from well-designed [CT and MRI] studies using sensitive methods…."[155]
2) Coffey is, at that very same time (mid-1994), saying that prospective studies are needed and,
3) There *was* credible evidence in

   a) Coffey et al., 1988, in which he states that one explanation of his evidence was that "…ECT may have caused cerebral atrophy…."[156]
   b) Coffey et al., 1991, reviewed above.
   c) Andreasen et al., 1990, which found a correlation between the number of previous ECT treatments and increased lateral ventricular volumes (loss of brain tissue) measured by MRI.[157]
   d) Dolan, RJ., et al., 1986, a CT scan study which found, "Patients with a past history of treatment by electroconvulsive therapy showed more sulcal widening in the parietal and occipital areas than those not so treated."[158] The authors explained that, "There was no evidence of association between the presence of these changes [sulcal widening in frontal, temporal, and interhemispheric areas of the brain] and a family history of depression, the duration of depressive illness, the age of onset of illness, the course of illness or exposure to psychotropic medication."[159] There was also no association with age or alcohol use. Coffey et al. 1991, in reviewing previous brain scan research, left this study out but did include a 1985 Dolan study which found no association between ventricular size and ECT.
   e) Calloway et al, 1981, which reported that frontal lobe atrophy was significantly more common in elderly depressed patients who had received ECT than in those who had not.[160]
   f) Weinberger et al., 1979, which found that cortical atrophy was significantly more common in schizophrenic patients who had received ECT than in those who did not.[161]

There were definite weaknesses in all the MRI studies. Many were retrospective. But that does not mean there is no credible evidence to support the idea that ECT causes structural changes. At best, this question has not been answered. Yet the 2001 APA task force report does not even discuss MRI results or the question of ECT and structural brain changes.

This, unfortunately, is typical of psychiatry's don't look, don't tell policy: Make a minimal effort to find evidence. Then, regardless of what is found, proclaim to the public that there is no evidence.

A Final Word

In 2002 ECT proponent Richard Abrams wrote a guest editorial for *The Journal of ECT* entitled, "Does Brief-Pulse ECT Cause Persistent or Permanent Memory Impairment?"[162] His piece demonstrates how psychiatry's don't look, don't tell policy perpetuates itself.

Abrams criticizes the results of Weiner's 1986 study, which found significant autobiographical memory loss at six months, for lack of peer review and the omission of "important methodological details," including "the actual dosages administered." However, a careful reading of Weiner's study shows that he calculated "a variety of electrical parameters," including stimulus intensity in Joules and coulombs, for each treatment condition.[163] Abrams apparently made no effort to obtain that data from Weiner, preferring to complain that the data was not available in published articles. (Perhaps they are simply not on speaking terms. Abrams and Weiner work for competing shock machine manufacturers.) Significantly, Weiner's 1986 study may well be one of the few autobiographical memory studies in which patients received the type of ultrathreshold doses that have never been formally studied but are delivered to many patients today.

In any event, Abrams fails to cast his critical eye on Calev's 1991 study, which, as we reviewed above, was riddled with suspect conclusions and methodology. He simply states that Calev was "unable to confirm the results reported by Weiner…."

Abrams then proceeds to misstate the results of Sackeim's 1993 and 2000 studies of autobiographical memory. According to Abrams, neither study demonstrated significant autobiographical memory impairment at 2 months relative to baseline. In fact, Sackeim did not publish the results of autobiographical memory testing at two months in either of those studies, so we don't really know exactly what level of impairment occurred. But in his 1993 study Sackeim did state, "Scores [at the two-month follow-up] on the retrograde Autobiographical Memory Interview [AMI] indicated no change in the consistency of recall relative to the post-treatment value."[164] Those post-treatment AMI scores demonstrated significant memory impairment. And in his 2000 study Sackeim wrote, "Two months after ECT, retrograde amnestic deficits were greatest among patients treated with BL ECT."[165]

McElhiney and Sobin, as we have seen, did report autobiographical memory impairment at two-months. So, of course, they are not mentioned in Abrams' editorial. Moreover, in all of

these studies we continue to miss the focus on individual responses that Weiner called for in 1984.

Finding (in his superficial analysis of 5 studies) no evidence of autobiographical memory deficits at two months, Abrams then presents what he apparently imagines to be his rhetorical coup de grâce: "How, then, to account for the fervently expressed assertion in a recent editorial by Dr. Harold Sackeim that, following ECT, *'virtually all patients experience some degree of persistent and, likely, permanent retrograde amnesia'*?"[166] (author's emphasis)

The answer is simple: Because virtually all patients do. Sackeim's deposition testimony adds weight to this conclusion. Two-thirds of his audience of ECT practitioners raised their hands when asked if they had observed "long term negative effects" of ECT.

So when shock practitioners claim that there is no evidence of ECT producing autobiographical memory loss, they do so only because,

1) the proper studies have never been undertaken;
2) they are denying or misrepresenting the outcomes of research that has been completed; or
3) they are denying or misrepresenting the outcomes of their own practice. Psychiatrists who witness long term memory loss in their patients are surely responsible for sharing their own experience, regardless of whether it is mentioned in psychiatric journals.

Unfortunately this minimizing of the effects of psychiatric treatment reflects a pattern that has been repeated all too often in the field of psychiatry. It can take decades for what is known by practicing psychiatrists to be confessed (or revealed through lawsuits and Freedom of Information Act requests) to the public at large. The movement disorders produced by antipsychotic drugs (e.g., tardive dyskinesia and dystonia) and the suicidal ideation and agitation produced by SSRI antidepressant drugs are two obvious examples.

Walking hand in hand with the policy of minimizing negative effects is the policy of exaggerating effectiveness, which, as we have seen, is characteristic of psychiatry's promotion of ECT.

A basic principal of medicine, the principal of informed consent, is missing in psychiatry. This is certainly the case with what we have politely referred to here by psychiatry's chosen name, electroconvulsive therapy. It should be evident now that this practice does not deserve its euphemistic title. The brain is being shocked. The result is a grand mal seizure, memory loss, and brain damage.

Psychiatry has not been simply negligent in researching and reporting these *primary* effects. It has pursued a policy designed to lead away from the truth, often avoiding study designs that might produce results unfavorable to shock. It has made false claims unsupported by published

research. It has deliberately deceived. It has looked away and remained silent while millions have been harmed.

References

1. Elliot Valenstein, *Blaming the Brain*, New York, The Free Press, 1998, p. 3.
2. Joseph Glenmullen, *Prozac Backlash*, New York, Simon and Schuster, 2000, p. 196.
3. U.S. Department of Health and Human Services. *Mental Health: A Report of the Surgeon General—Executive Summary*. Rockville, MD: U.S. Department of Health and Human Services, Substance Abuse and Mental Health Services Administration, Center for Mental Health Services, National Institutes of Health, National Institute of Mental Health, 1999, p. 48.
4. Ibid., p. 44.
5. Steven Sharfstein, "All Fired Up," *People*, 11 July 2005, p. 87.
6. Robert J. Waldinger, *Psychiatry for Medical Students*, Third Edition, American Psychiatric Press, 1997, p. 567.
7. Prudic, J., Sackeim, H., Devanand, D.P., "Medication Resistance and Clinical Response to Electroconvulsive Therapy," *Psychiatry Research*, 31:287-296, 1990.
8. Prudic, J., Haskett RF, Julsant B, et al., "Resistance to antidepressant medications and short-term clinical response to ECT, *American Journal of Psychiatry* 153:985-992, 1996.
9. Sackeim H., Prudic J., Devanand D.P., et al., "A Prospective Randomized Double-blind Comparison of Bilateral and Right Unilateral Electroconvulsive Therapy at Different Stimulus Intensities," *Archives of General Psychiatry*, 2000;57:425-434.
10. Prudic, J., Olfson, M., Marcus, S., et al., "Effectiveness of Electroconvulsive Therapy in Community Settings," *Biological Psychiatry* 2004;55:301-312.
11. American Psychiatric Association, *The Practice of Electroconvulsive Therapy, Recommendations for Treatment, Training, and Privileging*, Second Edition, 2001, p. 10.
12. *The National Institute of Mental Health (NIMH) recently completed the Sequenced Treatment Alternatives to Relieve Depression (STAR-D) study, a two-stage, 28-week antidepressant treatment study. During the first stage 2,876 generally moderately depressed patients were treated with high doses of the SSRI antidepressant Celexa. Symptoms remitted in only 790 (27.5%) patients. During the second stage one group switched to a second antidepressant, also at high doses and frequently augmented with anti-anxiety or sedative drugs.   Approximately one in four in this group remitted. [See Rush, Trivedi, et al., "Bupropion-SR, Sertraline, or Venlafaxine-XR after Failure of SSRIs for Depression," *The New England Journal of Medicine*, 354;12, March 23, 2006, p. 1231.] Another group of patients who had their Celexa augmented with a second drug did slightly better (about 30% remitted). [See Trivedi, Fava, et al., "Medication Augmentation after the Failure of SSRIs for Depression," *The New England Journal of Medicine*, 354;12, March 23, 2006, p. 1243.] Overall approximately 50% of patients remitted over the 28 weeks.

    However, the study excluded anyone who had previously not responded to one of the study medications. Therefore, the study population was not a true random sample. We can only guess what the remission rates would have been for a random sample of patients who had never taken any of the antidepressants used in this study – surely closer to 40%.

    Moreover, a continued follow-up of those who had achieved remission would undoubtedly have found that treatment began to fail a significant percentage of them. In his book, *Prozac Backlash*, Harvard psychiatrist Joseph Glenmullen wrote that systematic studies have confirmed that SSRI antidepressants "wear off in at least 30-40% of patients." (p. 91) The studies to which Glenmullen referred were year long studies.

    Thus, STAR-D's 28 week, 50% remission rate, which probably represents a rate closer to 40% in a randomly selected population, would be closer to 25-30% after another 6 – 12 months, with patients having tried at least three different antidepressants – at high doses. In short, "medication resistance," which really means treatment failure, is the rule, not the exception. A 2005 summary of antidepressant studies in the

British Medical Journal concluded, "Recent meta-analyses show selective serotonin reuptake inhibitors [SSRIs] have no clinically meaningful advantage over placebo." [Joanna Moncrief and Irving Kirsch, "Efficacy of Antidepressants in Adults," *British Medical Journal* 2005, 331, 16 July 2005, 155-157.]

13. Prudic et al., 1990, op. cit., p. 292..
14. Prudic et al., 1996, op. cit., p. 989.
15. American Psychiatric Association, 2001, op. cit., p. 7.
16. Husain et al., ""What Do Psychiatrists Mean by Medication Resistance as an Indication for Electroconvulsive Therapy? *Journal of ECT*, Vol. 21, No. 4, December 2005, p. 211-213.
17. From the webpage of the the University of North Carolina (Chapel Hill) Department of Psychiatry (http://www.psychiatry.unc.edu/clinicalservices/ect.htm).
18. From the webpage of Northern County Psychiatric Associates, Baltimore, Maryland (http://www.ncpamd.com/Adult_Depression.htm).
19. From Emory Healthcare, affiliated with the Emory University School of Medicine and Emory University Hospital (http://www.emoryhealthcare.org/departments/fuqua/patient_info/Electroconvulsive_Th.html).
20. From Businessweek.com (http://www.businessweek.com/magazine/content/05_18/b3931124_mz070.htm).
21. Quote from the website of the American Psychiatric Association: http://www.psych.org/research/apire/training_fund/clin_res/index.cfm.
22. Prudic, J., Olfson, M., Marcus, S., et al., "Effectiveness of Electroconvulsive Therapy in Community Settings," *Biological Psychiatry* 2004;55:301-312, p. 301.
23. Ibid., p. 301.
24. Ibid., p.304.
25. Ibid.
26. Ibid.
27. Ibid., p. 310.
28. Ibid., p. 309.
29. Prudic, J., Sackeim, H., Devanand, D.P., "Medication Resistance and Clinical Response to Electroconvulsive Therapy," *Psychiatry Research*, 31:287-296, 1990.
30. Ibid., p. 294.
31. Sackeim et al., "The Impact of Medication Resistance and Continuation Pharmacotherapy on Relapse following Response to Electroconvulsive Therapy in Major Depression," *Journal of Clinical Psychopharmacology*, Vol 10, No. 2, April 1990, 96-104.
32. Devanand, Sackeim and Prudic, "Electroconvulsive Therapy in the Treatment-Resistant Patient," *Psychiatric Clinics of North America*, Vol. 14, No. 4, December 1991, 905-923, p. 916.
33. Sackeim et al., "Continuation Pharmacotherapy in the Prevention of Relapse Following Electroconvulsive Therapy," *JAMA*, March 14, 2001, Vol. 285, No.10, 1299-1307.
34. Ibid., p.1304.
35. Ibid., p.1299.
36. Ibid., p. 1300
37. Ibid.
38. Ibid., p.1305.
39. American Psychiatric Association, 2001, op. cit., p. 10.
40. Abraham KR, Kulhara P, "The Efficacy of electroconvulsive therapy in the treatment of schizophrenia: a comparative study," *British Journal of Psychiatry* 151:152-155, 1987, p. 152. Brandon S, Cowley P, McDonald C, et al., "Leicester ECT trial: Results in Schizophrenia," *British Journal of Psychiatry* (1985), 146, 177-183, p. 177.
41. Brandon, op. cit., p. 180 (figure 4).
42. Taylor, P, Fleminger, JJ, "ECT for Schizphrenia," *The Lancet* 1, 1380-1382, June 28, 1980.
43. Brandon S., Cowley P., McDonald C., et al., "Electroconvulsive therapy: results in depressive illness from the Leicestershire trial," *British Medical Journal*, Volume 288, 7 January 1984, 22-25, p. 22.
44. Krystal et al, including Weiner, "EEG Effects of ECT: Implications for rTMS," *Depression and Anxiety* 12:157-165 (2000), p. 160.
45. Sackeim, Luber, Moeller et al., "Electrophysiological Correlates of the Adverse Cognitive Effects of Electroconvulsive Therapy," *The Journal of ECT*, 16(2): 110-120, 2000, p. 110.
46. Ibid., p. 111.
47. Ibid. p. 115.

48. Ibid., p. 116.
49. Sobin, Sackeim, et al., "Predictors of Retrograde Amnesia Following ECT," *Am J Psychiatry*, 152:7, 995-1001, July 1995.
50. Sackeim, Luber, Moeller, op. cit., p. 117.
51. Krystal (2000), op. cit., p.161.
52. Ibid., p. 160.
53. Sackeim, Prudic, Devanand et al., "A Prospective, Randomized, Double-blind Comparison of Bilateral and Right Unilateral Electroconvulsive Therapy at Different Stimulus Intensities," *Arch Gen Psychiatry*, Vol. 57, May 2000, p. 425.
54. Sackeim, "Central Issues Regarding the Mechanisms of Action of Electroconvulsive Therapy: Directions for Future Research," *Psychopharmcology Bulletin*, Vol. 30, No. 3, 1994, p. 296.
55. McCall, Rebousin, Weiner, et al., "Titrated Moderately Suprathreshold vs Fixed High-Dose right Unilateral Electroconvulsive Therapy," *Arch Gen Psychiatry*, Vol. 57, May 2000, p. 438.
56. Weiner, Richard D., "Does electroconvulsive therapy cause brain damage?" *The Behavioral and Brain Sciences* (1984) 7, 1-53, p. 9.
57. Michael Alan Taylor, *The Fundamentals of Clinical Neuropsychiatry*, Oxford University Press, 1999, p. 223.
58. Sackeim, Luber, Moeller, op. cit., p. 116-117.
59. Ibid., p.114.
60. Sobin, Sackeim, et al., "Predictors of Retrograde Amnesia Following ECT," *Am J Psychiatry*, 152:7, 995-1001, July 1995, p. 998.
61. Ibid., p.999.
62. The UK ECT Review Group, "Efficacy and safety of electroconvulsive therapy in depressive disorders: a systematic review and meta-analysis," *The Lancet*, Vol. 361, March 8, 2003, p. 806.
63. Douglas G. Cameron, "ECT: Sham Statistics, the Myth of Convulsive Therapy, and the Case for Consumer Misinformation," *The Journal of Mind and Behavior*, Winter and Spring 1994, Vol. 15, Nos. 1 and 2, pp. 177- 198.
64. Krystal et al., including Weiner, "ECT Stimulus Intensity: Are Present ECT Devices Too Limited? *American Journal of Psychiatry*; June 2000; 157(6), p. 963.
65. Sackeim, Luber, Moeller, op. cit., p. 114.
66. Ibid., p.112.
67. Sackeim, "Central Issues Regarding the Mechanisms of Action of Electroconvulsive Therapy: Directions for Future Research," *Psychopharmcology Bulletin*, Vol. 30, No. 3, 1994, p. 294.
68. American Psychiatric Association, 2001, op. cit., p. 208.
69. Ibid., p. 71.
70. American Psychiatric Association, *The Practice of Electroconvulsive Therapy, Recommendations for Treatment, Training, and Privileging*, 1990, p. 61.
71. Freeman, C.P.L. and Kendell, R.E., "Patients' Experiences of and Attitudes to Electroconvulsive Therapy," *Ann NY Acad Sci* 462:341-353, 1986, p. 351.
72. Ibid., p. 344.
73. Ibid., p. 346.
74. Ibid., p. 351.
75. Weeks D., Freeman CP, Kendell RE, "ECT: III: Enduring Cognitive Deficits?" *British Journal of Psychiatry* 137: 26-37, 1980, p.34.
76. Squire, LR, "Memory Functions as Affected by Electroconvulsive Therapy," *Ann NY Acad Sci* 462:307-314, 1986, p. 311.
77. Squire, et al., "Memory Complaint After Electroconvulsive Therapy: Assessment with a New Self-rating Instrument," *Biological Psychiatry*, Vol. 14, No. 5, 1979:791-801, p. 792.
78. Squire and Chace, "Memory Functions Six to Nine Months After Electroconvulsive Therapy," *Archives of General Psychiatry*, Vol 32, Dec 1975, 1557-1564, p.1562.
79. Squire and Slater, "Electroconvulsive Therapy and Complaints of Memory Dysfunction: A Prospective Three-year Follow-Up Study," *British Journal of Psychi*atry (1983), 142, 1-8, p. 5.
80. Diana Rose, Pete Fleischmann, Til Wykes, Morven Leese and Jonathan Bindman, "Patients' perspectives on electroconvulsive therapy: systematic review," *British Medical Journal*, Volume 326, 21 June 2003, p. 1365.

81. Ibid., p. 1366.
82. Squire 1986, p. 310.
83. Id.
84. Id.
85. Weiner, Richard D., "Does electroconvulsive therapy cause brain damage?" *The Behavioral and Brain Sciences* (1984) 7, 1-53, p. 13.
86. Squire, Slater, Miller, "Retrograde Amnesia and Bilateral Electroconvulsive Therapy," *Archives of General Psychiatry*, Vol 38, Jan 1981, 89-95.
87. APA, 1990, op. cit., p. 61.
88. Weiner, Richard D., et al, "Effects of stimulus parameters on cognitive side effects," *Ann N Y Acad Sci*, 462:315-325, 1986, p. 321.
89. Id.
90. Id.
91. American Psychiatric Association, *The Practice of Electroconvulsive Therapy, Recommendations for Treatment, Training, and Privileging*, Second Edition, 2001, p. 71.
92. Videotape depostion of Harold Sackeim, Ph.D., Jamaica, New York, March 14, 2004, 10:00 a.m. In the case of Akkerman v. Johnson, Court of the State of California for the County of Santa Barbara, Anacapa Division, Case No. 01069713, p. 147
93. Ibid., p. 148-149.
94. Ibid., p. 150-151.
95. Coffey, C. Edward, Cummings, Jeffrey L, Duffy, James D, Fink, Max, et.al., "Assessment of Treatment Outcomes in Neuropsychiatry: A Report From the Committee on Research of the American Neuropsychiatric Association," *Journal of Neuropsychiatry and Clinical Neurosciences*, Vol. 7(3) Sum 1995, p. 287.
96. Ibid., p. 289.
97. Coffey, Edward C., "The Institute of Medicine's "Quality Chasm" Report: Implications for ECT Care," *The Journal of ECT*, 19 (1): 1-3, 2003.
98. Weiner 1984, p. 15.
99. Ibid., pp. 15-16.
100. Squire and Slater 1983; Weiner et al., 1986; Sackeim, HA, Prudic J., Devanand DP., et al., "Effects of stimulus intensity and electrode placement on the efficacy and cognitive effects of electroconvulsive therapy," *New England Journal of Medicine*, 1993;328:839-846; Sackeim, Prudic, Devanand, et al., "A Prospective, Randomized, Double-blind Comparison of Bilateral and Right Unilateral Electroconvulsive Therapy at Different Stimulus Intensities," *Archives of General Psychiatry*, Vol 57, May 2000, 425-434.
101. Sackeim Deposition (see footnote #59), pp. 230-231.
102. Squire LR (b), "Memory functions as affected by electroconvulsive therapy," *Ann N Y Acad Sci* 462:307-314, 1986, p. 310.
103. Frith et al, "A comparison of some retrograde and anterograde effects of electroconvulsive shock in patients with severe depression," Br. J Psychol. 1987;78:53-63, p. 54.
104. Ibid., p. 56.
105. Calev et al., "Early and long-term effects of electroconvulsive therapy and depression on memory and other cognitive functions," *Journal of Nervous and Mental Disease*, Vol 179(9), 526-533, Sept 1991, p. 529.
106. Ibid., p. 527. (Abrams confirms that it was 1.5 times threshold, not 150% above threshold, in a 2002 editorial in *The Journal of ECT*, Vol. 18 (2) June 2002, pp. 71-73.
107. Ibid., p. 530.
108. Ibid., p. 531. According to Calev, the personal memory testing data, "…shows no evidence for an amnestic time gradient."
109. Ibid., p. 526.
110. Lisanby et al., "The effects of electroconvulsive therapy on memory of autobiographical and public events," *Arch Gen Psychiatry* 57:581-590, 2000, p. 589.
111. APA, 2001, op. cit., p. 70.
112. Calev, op. cit., p. 531.
113. Coleman, Sackeim et al., "Subjective Memory Complaints Prior to and Following Electroconvulsive Therapy," *Biological Psychiatry*, 39:346-356, 1996, p. 348. Coleman writes, "The sample included 70 of

the 96 patients who had participated in a double-blind, random assignment study of the effects of electrode placement and stimulus dosage on the efficacy and side effects of ECT (Sackeim et al 1993b)."

114. McElhiney et al., "Autobiographical memory and mood: effects of electroconvulsive therapy," *Neuropsychology* 9:501-517, 1995. McElhiney writes, "In the larger parent study, the treatment groups differed in the extent of short-term clinical improvement (Sackeim et al., 1993). Low-dosage RUL ECT lacked efficacy, high-dosage RUL ECT was intermediate, and both forms of BL ECT were equivalent and superior to either form of RUL ECT. A similar pattern held in the subsample included here." (p. 508)

115. Richard Abrams, "Does Brief-Pulse ECT Cause Persistent or Permanent Memory Impairment?" *The Journal of ECT*, Volume 18(2) June 2002, pp. 71-73.

116. Sackeim et al.(b), "Effects of Stimulus Intensity and Electrode Placement on the Efficacy and Cognitive Effects of Electroconvulsive Therapy," *The New England Journal of Medicine*, 328:839-846, 1993, p. 844 (Table 5). The low-dose bilateral group did even worse, with consistencies at 66±11.

117. McElhiney et al., "Autobiographical memory and mood: effects of electroconvulsive therapy," *Neuropsychology* 9:501-517, 1995. Remote memory consistencies for the No-crossover group (n=19) were 49.4 with an SD of 19.4. Consistencies for the crossover group were 47.1 with an SD of 12.3.

118. Sobin, Sackeim, et al., "Predictors of Retrograde Amnesia Following ECT," *Am J Psychiatry*, 152:7, 995-1001, July 1995, p. 995.

119. Sackeim et al.(c), "A Prospective, Randomized, Double-bind Comparison of Bilateral and Right Unilateral Electroconvulsive Therapy at Different Stimulus Intensities," *Arch Gen Psychiatry*, Vol 57, 425-434, May 2000, p. 433.

120. Sackeim 1993, Table 2 (p. 842) shows 27 patients received high dose bilateral ECT. He also states, "Of the 40[out of 96] patients who did not respond to therapy and were not randomly assigned to high-dose bilateral therapy, 34 (85%) completed the crossover phase." (p. 842). Sobin's 71 patients were "randomly assigned to four treatment groups," (p. 996) indicating that approximately 18 may have received high dose bilateral treatment in the randomized phase. It is unclear how many subjects received crossover treatment. We know at least 28 patients received only one course of ECT (p. 998). "Sobin writes, "45 patients completed the Autobiographical Memory Interview at all three time points [before treatment, 1 week post-ECT, 2 months post-ECT]. Seventeen of these patients had received a second, crossover course of ECT following the randomized phase." (p. 997) All of this suggests that there were at least 17 crossover patients and 28 who received one course of ECT (= 45), with possibly 18 of the 28 having received high-dose BL. In the Sackeim et al.2000 study, 20 patients were initially assigned to the high dose bilateral condition and 36 patients received crossover treatment. (p. 429).

121. Weiner's long-term personal memory results included 9 pulse bilateral subjects and 11 sine wave bilateral subjects. In Sackeim et al., 1993, there were15 high dose bilateral patients and 27 crossover patients among the 70 patients who were followed for 1 year after ECT (Figure 2, p. 843). In Sackeim et al. 2000, of the 55 patients who participated in the 2-month follow-up, 10 had received a single course of bilateral treatment and 25 had received bilateral as a crossover treatment. (p. 530) Therefore, at most, 35 of the 2-month follow-up patients had received high dose bilateral treatment. Some may actually have received low-dose bilateral. Sobin's high-dose bilateral figures are explained in footnote 95.

122. Ibid., p. 432.

123. Krystal et al., including Weiner, "ECT Stimulus Intensity: Are Present ECT Devices Too Limited?" *American Journal of Psychiatry*; June 2000; 157(6), p. 963.

124. Sackeim et al., "Stimulus Intensity, Seizure Threshold, and Seizure Duration: Impact on the Efficacy and safety of Electroconvulsive Therapy," *Psychiatric Clinics of North America*, Vol. 14, No. 4, December 1991, p. 809.

125. McCall, Reboussin, Weiner, et al., "Titrated Moderately Suprathreshold vs Fixed High-Dose Right Unilateral Electroconvulsive Therapy," *Arch Gen Psychiatry,* Vol. 57 No. 5, May 2000, 438-444.

126. American Psychiatric Association, op. cit., p. 158.

127. Sackeim et al. 1991, op. cit., p. 807.

128. Ibid., p. 806.

129. Ibid., pp. 806-807.

130. Ibid., p. 807.

131. McCall et al., including Weiner, "Titrated Moderately Suprathreshold vs. Fixed High-Dose Right Unilateral Electroconvulsive Therapy," *Arch Gen Psychiatry*, Vol 57, May 2000, p. 438.

132. Sackeim et al. 1991, op. cit., p. 810.
133. Sackeim et al., "Studies of Dosage, Seizure Threshold, and Seizure Duration in ECT," *Biological Psychiatry*, 1987; 22:249-268.
134. Sackeim et al., "Seizure Threshold in Electroconvulsive Therapy, Effects of Sex, Age, Electrode Placement, and Number of Treatments," *Arch Gen Psychiatry*, Vol. 44, April 1987, p. 358.
135. Coffey et al., including Weiner, "Seizure Threshold in Electroconvulsive Therapy: I. Initial Seizure Threshold," *Biological Psychiatry* 1995;37:713-720, p. 716.
136. American Psychiatric Association, op. cit., p. 160.
137. Ibid., p. 159.
138. Rasmussen, "Stimulus Titration and ECT Dosing:Commentary," *The Journal of ECT*, Volume 18(1), March 2002, pp. 10-11.
139. Coffey, Lucke, Weiner, et al., "Seizure Threshold in Electroconvulsive Therapy (ECT) II. The Anticonvulsant Effect of ECT," *Biological Psychiatry*, 1995;37:777-788. Figure 1 on p. 783.
140. Ibid., Table 3 on page 782.
141. Michael Alan Taylor, *The Fundamentals of Clinical Neuropsychiatry*, Oxford University Press, 1999, p. 218.
142. Edward Coffey, Richard Weiner et al., "Brain anatomic effects of electroconvulsive therapy: A prospective magnetic resonance imaging study," *Archives of General Psychiatry*," Vol 48(11) Nov 1991, 1013-1021.
143. Ibid., p. 1014, Table 1.
144. Ibid., p. 1015.
145. Ibid.
146. Ibid.
147. Ibid., p. 1014.
148. Ibid.
149. Ibid., p. 1013.
150. Ibid., p. 1018.
151. Ibid.
152. Ibid. p. 1019.
153. Coffey, "The Role of Structural Brain Imaging in ECT," *Psychopharmacology Bulletin* 30(3): 477-483, 1994, p. 478.
154. Devanand, Dwork, Hutchinson, "Does ECT Alter Brain Structure?" *American Journal of Psychiatry*, 151:7, July 1994, p. 957-970.
155. Ibid., p. 960.
156. Coffey et al., "Effects of ECT on Brain Structure: A Pilot Prospective Magnetic Resonance Imaging Study," *American Journal of Psychiatry*, 145:6, 701-704, June 1988, p. 704.
157. Andreasen, et al., "MRI of the Brain in Schizophrenia," *Arch Gen Psych* 1990;47:35-41.
158. Dolan, RJ et al., "The cerebral cortical appearance in depressed subjects," *Psychological Medicine*, 1986, 16, 775-779.
159. Ibid., p. 778.
160. Calloway SP, et al., "ECT and cerebral atrophy: a computed tomographic study," *Acta Psychiatrica Scandanavia*," 1981;64:442-445.
161. Weinberger, et al, "Structural abnormalities in the cerebral cortex of chronic schizophrenic patients," *Arch Gen Psych* 1979;36:935-939.
162. Richard Abrams, "Does Brief-Pulse ECT Cause Persistent or Permanent Memory Impairment," *The Journal of ECT*, Vol. 18(2), June 2002, pp. 71-73.
163. Weiner, Richard D., et al, "Effects of stimulus parameters on cognitive side effects," *Ann N Y Acad Sci*, 462:315-325, 1986, p. 321. On page 317 Weiner states, "Measures of stimulus intensity showed highly significant intergroup differences with respect to stimulus waveform (p < 0.0001), with sine-wave stimuli associated with 2.6 times the stimulus energy (Joules), 3.1 times the applied charge (coulombs), and 6.9 times the mean current (coulombs per second) as that associated with pulse stimuli."
164. Sackeim et al., 1993, op. cit., p. 843-844.
165. Sackeim et al., 2000, op. cit., p. 425.
166. Abrams 2002, op. cit., p. 72.